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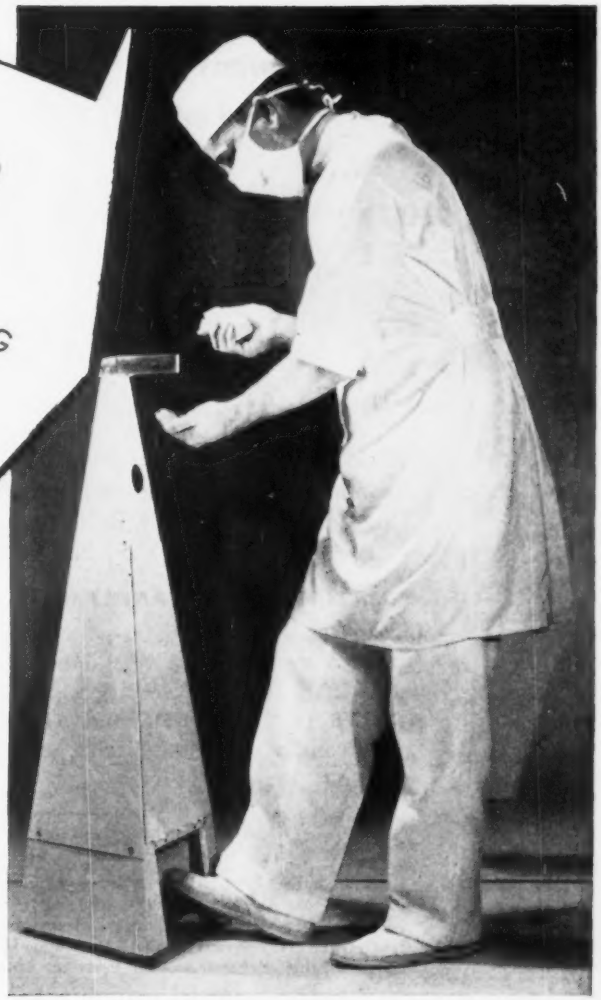

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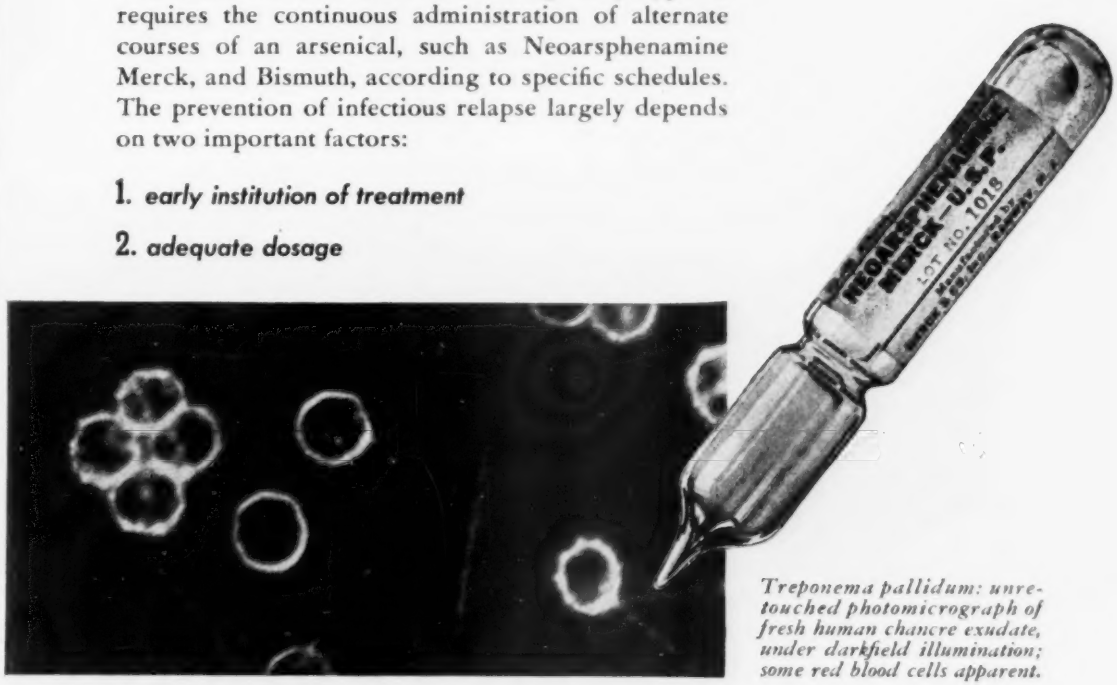
2. *adequate dosage*



Treponema pallidum: unretouched photomicrograph of fresh human chancre exudate, under darkfield illumination; some red blood cells apparent.

The statistical studies of Padget* have been of considerable value in establishing the need for treating primary syphilis during the seronegative stage. Under the best conditions obtainable, the syphilitic patient has less chance of being cured when treatment is delayed until the blood becomes seropositive than he has if therapy is instituted while it still is seronegative. Furthermore, the incidence of neurosyphilis has been found to be more than twice as high in seropositive primary syphilis cases than in any other.

Inadequate dosage of Neoarsphenamine will only lead to progression and extension of the syphilitic lesions. Low dosage will neither eradicate the infection nor permit the host to develop natural resistance to it. The incidence of infectious recurrence will be reduced to a minimum if treatment is continuous rather than intermittent, and provided that during the selected course of treatment, the patient at no time is without the influence of either Neoarsphenamine or Bismuth.

The first three months of treatment are most important in preventing future relapse. Negative serologic reactions do not necessarily indicate noninfectiousness; hence, patients should not be permitted to relax precautions or suspend therapy until the proper number of alternating, uninterrupted courses of Neoarsphenamine Merck and Bismuth Subsalicylate have been given.

*Padget, P., *Long-term results in treatment of early syphilis*, J. A. M. A. 116:7-11, 1941.

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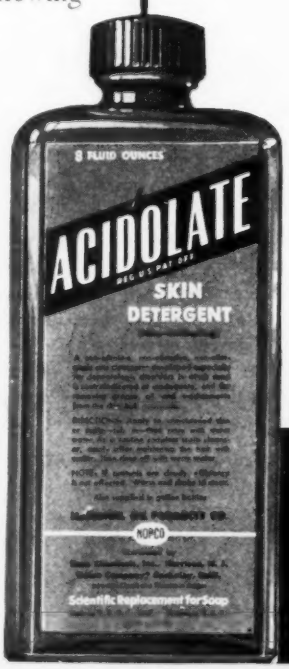

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**Meyer, E., and Arnold, L., Amer. J. Digest. Dis., 5:418.*

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- Cheilitis glandularis
- Chromidrosis
- Clavus
- Condylomata acuminata
- Cornu
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- Dermatitis herpetiformis
- Dermatitis (infectious eczematoid)
- Dermatitis papillaris capillitii
- Dermatitis seborrhoeica
- Dermatitis venenata
- Dermatophytosis
- Dermatophytide
- Eczema
- Epithelioma (basal cell)
- Epithelioma (prickle-cell)
- Epithelioma (multiple benign)
- Erysipelas
- Erythema induratum
- Favus (of the scalp)
- Folliculitis decalvans
- Furunculosis
- Granuloma fungoides
- Granuloma pyogenicum
- Granulosis rubra nasi
- Hodgkin's disease
- Hydrocystoma
- Hyperidrosis
- Hyperkeratosis of palms
- Hypertrichosis
- Impetigo
- Intertrigo
- Keloid
- Keratosis (arsenical)
- Keratosis follicularis
- Keratosis seborrheica
- Keratosis senilis
- Kraurosis vulvae
- Lepra nodosa
- Leukemia cutis
- Leukoplakia
- Lichen chronicus (Vidal)
- Lichen nitidus
- Lichen planus
- Lichen scrofulosorum
- Lichenification
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- Lupus miliaris disseminatus
- Lupus vulgaris
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- Lymphangioma circumscriptum
- Lymphangitis (chronic or recurrent streptococcic)
- Molluscum contagiosum
- Mycosis fungiodes
- Neurodermatitis
- Nervux pilosus
- Onychomycosis
- Papilloma
- Parapsodiasis lichenoides
- Paranychia
- Pernio
- Pityriasis rosea
- Pompholyx
- Pityriasis rosea
- Pompholyx
- Prurigo
- Pruritus
- Psoriasis
- Rhinophyma
- Rhinoscleroma
- Rosacea
- Sarcoid
- Sarcoma (general types)
- Sarcoma (giant cell)
- Sarcoma (Kaposi)
- Scrofuloderma
- Sebaceous cyst
- Sporotrichosis
- Sycosis (lupoid)
- Sycosis vulgaris
- Synovial lesions of the skin
- Syringoma
- Tinea barbae
- Tinea capitis
- Trichophytosis profunda
- Tuberculide (various types)
- Tuberculosis orificialis
- Tuberculosis verrucosa cutis
- Verruca plantaris
- Verruca vulgaris
- Xeroderma pigmentosum (keratosis and epithelioma)

*From "Diseases of the Skin"—Dr. Geo. C. Andrews. "X-Ray and Radium in the Treatment of Diseases of the Skin"—Dr. Geo. M. MacKee.

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BRANCHES IN PRINCIPAL CITIES OF U. S. A. AND CANADA

Archives of Dermatology and Syphilology

VOLUME 51 JUNE 1945 NUMBER 6

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ECTHYMA CONTAGIOSUM IN MAN

DATA CONCERNING ITS INCIDENCE IN SEVERAL WESTERN STATES; REPORT OF A CASE

LYLE B. KINGERY, M.D., AND JOYLE DAHL, M.D.

PORTLAND, ORE.

In 1923, Aynaud [1] reported the results of his studies on ecthyma contagiosum. In this report he described experimental procedures definitely identifying the disease as due to a filtrable virus and apparently peculiar to sheep and goats. Following this early work, the results of similar studies have been published by Glover,[2] Boughton and Hardy,[3] Oppermann and Stümpke,[4] Marsh and Tunnicliff [5] and others. As a result of these investigations, there is now available a rather complete and detailed knowledge of the disease as it exists in the animals studied. This information may be summarized somewhat as follows:

(*a*) The disease is due to a specific filtrable virus.

(*b*) It is apparently peculiar to sheep, goats and man.

(*c*) It is known to exist throughout most of the wool-producing areas of the world.

(*d*) Where preventive measures are not employed, it may be characterized by a high annual incidence rate with impressive mortality in susceptible animals.

(*e*) It may, therefore, at times assume economic aspects of considerable importance.

(*f*) And, finally, it can be efficiently prevented by the use of a specific vaccine.

In spite of the established frequency and world-wide distribution of the disease in animals, there is available relatively limited information concerning its occurrence in man. A few isolated reports have been published from widely separated localities. These include the publications by Peterkin [6] from England; by Orbaneja and Robert [7] from France; by Oppermann and Stümpke [4] from Germany, and by Schoch [8] and Nomland [9] in this country. Dermatologic texts devote but little space to a description of the manifestations of the infection in man. One is more or less entitled to assume, therefore, either that the human disease is one of extreme rarity or that the general medical profession is unfamiliar with this particular clinical picture. The present study was undertaken for the purpose of recording the findings in an additional case of human infection in a locality from which it had not been previously reported; to present the total clinical picture as found by various previous observers, and to establish the fact that at least in this country it is a disease of greater frequency and wider distribution than available information has led us to believe.

REPORT OF CASE

History.—Mr. W. H., aged 34, a farm laborer, was referred to us for diagnosis and advice on Jan. 6, 1944. His history revealed that during the three weeks preceding the time of consultation he had been engaged in treating a band of sheep for so-called "fluke disease." This treatment necessitated the forcible opening of the sheep's mouth and the insertion of a large capsule in a manner that would compel the animal to swallow the medication. In the process his hands and

From the University of Oregon Medical School, Department of Dermatology and Syphilology.

Read at the Sixty-Fifth Annual Meeting of the American Dermatological Association, Inc., Chicago, June 21, 1944.

1. Aynaud, M.: La stomatite pustuleuse des ovins (chancre du mouton), Ann. Inst. Pasteur **37**:498 (May) 1923.

2. Glover, R. E.: Contagious Pustular Dermatitis of Sheep, J. Comp. Path. & Therap. **41**:316 (Dec.) 1928.

3. Boughton, I. B., and Hardy, W. T.: Contagious Ecthyma (Sore Mouth) of Sheep and Goats, J. Am. Vet. M. A. **85**:150 (Aug.) 1934.

4. Oppermann, T., and Stümpke, G.: Der Lippengrind (Ekthyma contagiosum) der Schafe und seine Übertragbarkeit auf den Menschen, Arch. f. Dermat. u. Syph. **176**:337, 1937.

5. Marsh, H., and Tunnicliff, E. A.: Stomatitis in Young Lambs Involving Actinomyces Necrophorus and the Virus of Contagious Ecthyma, J. Am. Vet. M. A. **91**:600 (Nov.) 1937.

6. Peterkin, G. A. G.: The Occurrence in Humans of Contagious Pustular Dermatitis of Sheep ("Orf"), Brit. J. Dermat. **49**:492 (Nov.) 1937.

7. Robert, P., and Orbaneja, G.: Trois cas de granulomes angiopappillomateux eruptifs infectieux, Ann. de dermat. et syph. **8**:45 (Jan.) 1937.

8. Schoch, A.: Sheep Pox Infection in Man, Arch. Dermat. & Syph. **39**:1040 (June) 1939.

9. Nomland, R.: Human Infection with Ecthyma Contagiosum, a Virus Disorder of Sheep, Arch. Dermat. & Syph. **42**:878 (Nov.) 1940.

Fig. 1.—Lesions as they occur in an infected animal.

Fig. 2.—Note papulovesicular and papular pustular lesions, some with umbilication.

Fig. 3.—Portion of section showing hemorrhagic vesicle. The biopsy report and the photomicrograph were secured from Professor Warren C. Hunter, of the Department of Pathology, University of Oregon Medical School.

fingers were frequently traumatized by the sharp edges of the sheep's teeth. He stated that a large percentage of the animals had "pustular growths and scabs" on their lips (fig. 1).

Several days after beginning this work the lesions for which he consulted us began to appear. The patient further described exactly similar lesions on the hands and fingers of 2 of his co-workers. His general medical history was negative.

Examination.—The general physical examination and ordinary laboratory work revealed nothing noteworthy. The dermatologic examination revealed three lesions on the right thumb and scattered similar lesions on the fingers of each hand. Each lesion was situated on a circular erythematous base averaging from 1 to approximately 2 cm. in diameter. The lesions consisted of rather firm papules, papulovesicular and pustular lesions with some suggesting umbilication and

masses of blood pigment, clumps of bacteria and nuclei of what could well be disintegrated leukocytes were at times caught. The cavity was filled with intact erythrocytes, together with a few granulocytes and lymphocytes. The rete mucosum was intact except for the cells that bordered immediately on the blood-filled cyst. Here the more exposed cells were becoming detached and showed evidences of necrosis. The corium was the seat of many dilated vascular channels, most of which were quite devoid of blood. These could be either lymphatics or blood capillaries. Some areas of the corium contained leukocytes in moderate numbers. Some of these were granulocytes but the majority appeared to be lymphocytes. In addition to leukocytes, there were many other cells in the corium. These were rather pale, ovoid to spindle shaped, and looked not unlike the endothelial cells that lined the vascular channels. Their exact nature is unknown but they

TABLE 1.—*Reported Cases of Human Infection*

Reported by		Source of Infection	Incubation Period	Type of Lesion	Distribution	Accompanying Symptoms	Duration
Peterkin	1	Contact with animal	Not given	Ulcer	Finger	None	4 weeks
	2	Contact with animal	Not given	Nodule, umbilicated bleb	Hand	Not given	3+ weeks
	3	?	5 days	Umbilicated pustule	Finger	Pain	Not given
	4	Contact with animal	Several days	Umbilicated pustule	Eyelid	Pruritus	4+ weeks
	5	Contact with animal	Not given	Umbilicated pustules	Thumb	Not given	2 weeks
Robert and Orbaneja	1	Contact with animal	Not given	Pustular, vesicular, papillomatous	Nose and temporal region	Adenopathy	Electrocoagulation
	2	Contact with animal	Not given	Pustular, vesicular, papillomatous	Nose	Adenopathy	Electrocoagulation
Oppermann and Stümpke	1	Needle puncture	Apparently blood stream infection with fever and joint pains; blood injected into sheep produced typical lesions				
	2	Contact with animal	Several days	Vesicles, pustules, ulceration	Hand	Not given	Not given
	3	Contact with animal	Not given	Small inflammatory macules and vesicles	Hand and neck	Inflammation, swelling, itching	Not given
Schoch		Contact with animal	3 days	Pustules	Wrist	Adenopathy	3 weeks
Nomland	1	Contact with vaccine	4 days	Bullae, vesicles	Fingers	Pain, redness	3 weeks
	2	Contact with animal	7 days	Vesicles, pustules	Fingers	Swelling, inflammation	Not given
Authors' case		Contact with animal	Days	Papular, vesicular, pustular	Fingers	Mild pain and itching	3-4 weeks

hemorrhage. There was local heat, and the patient described mild accompanying subjective symptoms. There was no adenitis and no mention made of constitutional manifestations. Because of the interesting clinical picture and history, Dr. Thomas Saunders was asked to see the case with us and assisted in its identification. As stated by Nomland,[9] the diagnosis is made by exclusion. The condition bears no resemblance to the ordinary vesicular and pustular diseases of the hands. At the same time, a recent vaccination, vaccinia and milkers' nodules can be readily excluded. Photographs (fig. 2) were made and biopsy material taken but we were not given time for further laboratory study. A week or so later the patient's physician informed us that the lesions had completely healed under applications of a mild mercurial cream.

Pathologic Report.—Microscopically (fig. 3) there was found to be splitting of the epidermis through the stratum granulosum leaving an outer wall consisting of partially and fully keratinized epithelium in which are presumed to be young fibrous connective tissue cells. No inclusion bodies were made out in the epithelial cells of sections stained in the usual manner with hematoxylin and eosin.

Pathologic Diagnosis.—The diagnosis was: intracutaneous hemorrhagic vesicle with focal subacute inflammation.

Ordinarily, the total clinical picture of a given disease entity is most accurately represented by a gradual accumulation of the observations of several individual investigators. With this in mind, a compilation of the findings included in previous reports, together with our own, is shown in table 1. From the information thus tabulated certain general statements may be made regarding the characteristics of the disease as recorded thus far.

They are as follows:

1. Human infection is apparently acquired by actual contact with infected animals or laboratory material. There are no proved cases of infection from working in stables or from contact with vegetation in which the animals graze.

Fig. 4.—Stippled areas showing distribution of lesions in reported cases.

2. The incubation period is short, apparently varying from a few days to one week.

3. The evolution of the disease and lesional types are consistent. The first manifestation is usually noted as one or more inflammatory elevations with rapid development of vesicles or bullae which may, or may not, show hemorrhage. There is a tendency for some lesions to develop umbilication, and the contents often become purulent. Occasional ulceration occurs, and in some cases the lesions have become papillomatous.

4. The sites of predilection, as would be expected, include the fingers, hands and wrists. The occasional occurrence on parts of the face could well result from contact with material carried by the fingers or nails.

5. Accompanying symptoms are not striking. They may be completely absent or may include mild swelling, pain and adenitis.

6. The disease may well be self limited. In all reported cases the lesions have disappeared in a matter of weeks under the mildest types of local symptomatic therapy.

7. Finally, while the information in table 1 emphasizes the limited number of cases of human infection on record, the widely separated sources of these reports suggest a possible incidence and distribution not ordinarily included in the conception of the disease as it is now known.

In order to obtain further information relative to the frequency and geographic distribution of the disease in this country, questionnaires were sent to various agricultural colleges and experimental stations. The states chosen represent an area producing approximately 60 per cent of the country's total wool output. It was presumed that information obtained from these sources might be accepted as applying with reasonable accuracy to sheep-growing sections in general. Obviously, the data thus obtained represent the observations of persons most interested in the disease as it occurs in animals. At the same time, it is this group of investigators who have contributed most to our knowledge of the disease and by reason of their activities have been in constant touch with large numbers of herders and employees in whom one would expect the greatest incidence of infection. Their observations are recorded in table 2 [10] and permit certain

TABLE 2.[10]—*Observations on Human Infection by State Laboratories and Experiment Stations*

State	Frequency	Source of Infection	Type of Lesion	Distribution	Accompanying Symptoms	Duration	Immunity
Washington	Occasional	Contact with animal	Not given	Hands	Not given	Several weeks	Not given
Oregon	Seen occasionally among handlers	Contact with animal	Not given	Hands	Pain	Not given	Not given
California	Seen occasionally among handlers	Contact with animal	Pustules	Fingers	Pain, swelling, inflammation	Several days	Not given
Idaho	Known to occur	Contact with animal	Not given	Not given	Not given	Not given	For life
Montana	Occasional among handlers	Contact with animal	Vesicles, pustules	Hands	Not given	Not given	Not given
Wyoming	Occasional among handlers	Contact with animal	Not given	Not given	Not given	Not given	Not given
Texas	Fairly common	Infected animals, laboratory material	Vesicles, pustules, ulcers	Fingers, hands, lips	Inflammation, itching	10 days	Presumably for life

10. Personal communications to the authors from E. E. Wegner, State College of Washington, Pullman, Wash.; J. N. Shaw, Oregon State College, Corvallis, Ore.; J. W. Britton, College of Agriculture, Davis,

(Footnote continued on next page)

further generalizations concerning human infection. They are as follows:

1. Apparently, one may assume the disease to be endemic in many localities, observations varying from "occasional" to "fairly common" in the states contacted.

2. Complete unanimity of opinion exists concerning the mode of transmission, i. e., contact with infected animals or with laboratory material.

3. The types of lesions seen, their sites of election, the accompanying symptoms and their duration, when observations were made, correspond to those noted in table 1.

4. Finally, one of the observers [2] who has contributed largely to the perfection of an effective vaccine suggests the development of a lasting immunity following one attack.

SUMMARY

A summation of the foregoing data would seem to permit a reasonably complete description of human ecthyma contagiosum and to emphasize certain characteristics of the disease not stressed in previous reports. Primarily, clinical and experimental observations have definitely established the cause and methods of acquisition. Further, the eruptive manifestations and accompanying symptoms are fairly consistent and run true to type. The disease may occur on the face as well as on the fingers, hands and wrists. Individual immunity after one attack is a distinct possibility. Finally, while the lack of disabling symptoms and ease of curability make for a professional interest that is largely academic, the disease should be recognized as a dermatosis occurring with a greater frequency and wider geographic distribution than previous investigations have led one to believe.

ABSTRACT OF DISCUSSION

DR. RUBEN NOMLAND, Iowa City: I wish to express my appreciation of the presentation of the paper on this disease which is apparently rare.

People who live in rural areas have eruptions that are unfamiliar to the urban dermatologist, and most of these have escaped expert reporting. Ecthyma contagiosum is one of these diseases. Dr. Kingery and Dr. Dahl have definitely shown that in sheep-raising areas this disease is rather common.

I believe that as time goes on more and more cases will be recognized. The clinical description that he has given is comparable to descriptions of cases reported in the literature, to Dr. Schoch's case and to the 2 that I saw.

The clinical lesion has a great resemblance to a smallpox vaccination with small pustular formation and finally, a rather large, umbilicated pustule with a surrounding zone of great inflammation, drying up and leaving a necrotic crust. This is, as nearly as I can tell, the course of most of the lesions. Occasionally papillomatous lesions are seen.

I think there is a great similarity between this disease and milkers' nodules, another virus disorder of animals which, also, in the United States has been only briefly described. Milkers' nodules, as far as I know, have been described in only one paper in the American literature, and still milkers' nodules are rather common in rural communities. Again, it is usually a trifling infection, with a self-limited course, and does not come to the attention of the expert who might be interested in reporting it. However, one sees milkers' nodules rather frequently in rural Iowa. In talking to observing general practitioners in rural areas, one learns that the disease is rather common and seldom requires expert care.

Ecthyma contagiosum is in the same category—a fairly common, minor, self-limited disease which occurs in those areas in which sheep are bred.

DR. JAMES W. JORDON, Buffalo: We have had 1 case of this condition in Buffalo. The disease probably did not originate in the state of New York. It occurred in a trainman who handled sheep that were coming on a train from the West. About five days later he had an eruption similar to that described in this paper.

Recently 3 cases of milkers' nodes were seen. As far as I know, we have never had a case of this infection in Buffalo before. All of these came from one section, around East Aurora, which is a suburb of Buffalo. All the patients were milkers working on dairy farms. All reported that the cattle they worked with had some sort of eruption on the udders. The infection usually started in an area of broken skin such as a scratch or some other traumatic lesion. There is a decided difference clinically between the two diseases. Ecthyma contagiosum is a bullous or pustular eruption and milkers' nodes are red, raised vesicular nodules that resemble pyogenic granulomas except that the surfaces of the nodules do not become eroded. They usually start with a single lesion and about this two or three other nodules appear in close proximity. Shortly after the appearance of the nodules on the skin, the regional lymph nodes enlarge. The nodes are rather large, varying in size from that of a hickory nut to that of a walnut. They are firm, not attached to the overlying skin or the underlying tissues. This disease is self limited and tends to clear up of its own accord in six to ten months.

DR. JOSEPH V. KLAUDER, Philadelphia: An interesting aspect of this disease is the possible use of vaccination to extend the therapeusis of diseases of the skin caused by viruses and those presumably caused by viruses, in much the same way as vaccination for smallpox is used.

Does Dr. Kingery think that vaccination for this disease would be any more hazardous than vaccination for smallpox?

DR. BEDFORD SHELMIRE, Dallas: An eruption of the hands the clinical appearance of which is identical with that of human ecthyma contagiosum is by no means rare in my practice. Dr. Jordon has ably described the lesions when he stated they resemble multiple smallpox vaccinations. In most instances persons affected

Calif.; Glenn C. Holm, University of Idaho, Moscow, Idaho; Hadleigh Marsh, Agricultural Experiment Station, Bozeman, Mont.; A. M. Lee, University of Wyoming, Laramie, Wyo.; I. B. Boughton, Agricultural College of Texas, Sonora, Texas.

with the disease gave a history of milking cows and invariably the cows were said to have had sores on the udders. In at least 2 instances I called in veterinarians who diagnosed the eruption on the cow udders as "cowpox." Therefore, I simply dismissed the diagnosis as cowpox contracted from handling infected udders of cows. Since many of the patients also handled sheep it is possible that the disease was contracted from these animals.

I had the privilege of seeing the case of sheep-pox described by Schoch. There was absolutely no doubt that this man had contracted his infection from sheep. The appearance of the eruption and its clinical benign course were identical with those of the eruption I had seen so frequently following the milking of cows with infected udders. My impression is that sheep-pox and cowpox are clinically identical in man.

Dr. Samuel Ayres Jr., Los Angeles: I would like to have Dr. Kingery comment on the differentiation between ecthyma contagiosum, which apparently is a distinct entity, and the disease that I think every one is familiar with and which is obviously common, just plain ecthyma. I personally have never seen one of the cases described here, and I think there might be confusion in some minds unless some differentiation was brought out.

Dr. Lyle B. Kingery, Portland, Ore.: Mr. Chairman, your question concerning vaccination has been satisfactorily answered by the research work carried on at the various experiment stations. That is, inasmuch as the vaccine is prepared from material from actual lesions, then used as a preventive in noninfected animals, one may assume its use is attended by no risk. Is that what you had in mind?

Dr. Joseph V. Klauder, Philadelphia: Yes.

Dr. Lyle B. Kingery: Dr. Shelmire's question, I believe, is answered by some of the correspondence I had with the head of the department of veterinary medicine at our own state university. In my first letter I referred to the disease as "cowpox" and was immediately put in my proper place by being told that bacteriologically and clinically the two diseases were distinct entities, having no relationship. Furthermore, in the case we have reported there was no history of contact with cattle.

Dr. Ayres has suggested a possible clinical similarity between ecthyma contagiosum and ordinary ecthyma. If one were to see examples of these two diseases at the same time the complete dissimilarity would be obvious. The former begins as an inflammatory papule on which there is rapidly superimposed a hemorrhagic vesicle or bulla, this gradually becoming secondarily infected and frequently showing central umbilication. The latter begins as a pustule and practically always develops into a crusted pyogenic ulcer. In addition, the two diseases differ as to distribution of lesions, history of exposure and duration.

In closing, I want to express my appreciation of this discussion.

Photographs by Marsh and Tunnicliff.

RECURRENT, RESISTANT VESICULAR ERUPTION OF THE HANDS

J. HARPER BLAISDELL, M.D., AND JACOB H. SWARTZ, M.D.
BOSTON

The purpose of this paper is to call attention to a chronic, recurrent, resistant vesicular eruption of the palms and fingers that is being seen with increasing frequency and that has certain characteristics that identify it as a definite entity. One and two-tenths per cent of cases seen in our private practice belong in this group.

The predisposing factor in the appearance of this disease is chronic irritation of the affected parts, either through occupation or from overstimulating treatment of a simple dermatitis. The majority of cases observed appear in those exposed to hazardous occupations, such as machine operators, porters and bakers. Housewives, with their exposure to soap and hot water in the washing of dishes and clothes, to wide extremes of heat and cold in the kitchen and to house dust, are frequently victims of this disease. Its occurrence in the protected office worker is much rarer, and the eruption in these cases is usually limited to much smaller areas than in other patients. The housewife and office worker often give a history of a duration extending as long as ten years, the attacks coming on several times a year. The persistent use of vigorous and stimulating treatment for simple inflammations of the palms and fingers predisposes to the disease in persons otherwise not subject to it.

The disease occurs on the sides of the fingers, on the palmar aspects of the hands and fingers and in the interspace webbing. The invasion of the backs of the hands or the knuckles occurs only as a secondary spread of a dermatitis from the interspaces. The feet are uninvolved. In a majority of the cases the patient is of the nervous type, with active sweating to the extent of drops of perspiration being present during examination. The skin is often habitually cold and clammy. The unilateral or bilateral character of the disease is entirely dependent on the extent of the initial dermatitis. It is noncontagious.

The primary lesion is a vesicle, varying in size from that of a pinhead to that of a small split pea. It is pearly and translucent and appears on the surface of the skin without producing any trace of surrounding inflammation. It may occur in small clusters 0.6 to 0.12 cm. in diameter, each of which contains pearly vesicles of varying size. The secondary lesion is a scale, the size of which depends on the size of the vesicle.

The clinical history is frequently one of an obvious and adequately explained industrial dermatitis of the hands, which through lack of attention or overtreatment has persisted for several weeks. As the frank signs of the dermatitis subside, the pearly vesicles appear along the edges of the dermatitis, and on occasion in the more or less normal skin adjacent to the outbreak. If secondary infection occurs, the surface of the skin becomes oozing, purulent and crusting; the primary vesicles are masked by this complication. Overstimulating treatment produces the signs of typical acute dermatitis.

With simple drying, soothing treatment the vesicles dry and the skin desquamates. If the vesicles have appeared at about the same time over a period of two or three days, the process of involution results in a relatively normal-appearing skin at the end of ten to fourteen days. If the attack is severe and prolonged, vesicles in all stages of progress appear at the same time.

This disease consists of a recurring series of attacks. In many patients these occur at regular intervals, the most frequent period being every twenty-six to twenty-eight days. At times the interval is as short as eighteen days. So far as can be determined there is no relation to any event such as menstruation. An observing patient can frequently predict the coming of a new attack by the burning and itching of the affected parts twelve to twenty-four hours before the new vesicles appear.

The course of the disease is extremely variable in length. It may disappear after a few months, but often lasts for a year and occasionally as long as two or three years in spite of any treatment of which we are aware.

In the industrial worker protection from the original hazard or a change in occupation may obviate the element of dermatitis, but it has no influence on the recurring vesiculation. There is no seasonal variation, and extreme cold or heat has no influence on its course other than the pro-

Read at the Sixty-Fifth Annual Meeting of the American Dermatological Association, Inc., Chicago, June 20, 1944.

duction of a complicating element of secondary dermatitis.

Treatment is symptomatic and completely unsatisfactory. Soothing, drying and mildly antiseptic treatment controls the presenting dermatitis and its possible secondary infection without trouble. For this purpose warm soaks of boric acid or of potassium permanganate in solutions of 1:5,000 have proved satisfactory. An exudative dermatitis yields to a 5 to 10 per cent crude coal tar ointment. So far in our experience, nothing has a specific influence on the recurring vesiculation. Use of roentgen and ultraviolet radiation gives temporary relief and hastens the involution of the given attack of vesiculation, but neither is curative. More recently we have used applications of aluminum chloride or hydrosol, and the resulting decrease of the excessive sweating seen in many cases seems promising, but this therapy has not been in use long enough to warrant evaluation.

In the cases studied direct microscopic examination of the roofs of the vesicles has not revealed fungus. The cutaneous scales planted on Sabouraud's medium and grown at room temperature have yielded no pathogenic fungous growth. Some scales yielded Staphylococcus albus, but the ratio was the same as that of normal scales of skin planted under the same conditions. A few scales showed a more lucrative growth.

The following cases serve to illustrate the clinical entity discussed.

REPORT OF CASES

Case 1.—Mrs. D., a 24 year old housewife, was first seen on Aug. 19, 1943, at which time there had been an eruption on her hands for four years. The eruption appeared during her last year in college coincidentally with a course in chemistry and became worse during the first two years of her married life. It appeared three or four times each year for periods of several weeks. During the ten months previous to the initial visit the outbreak had been continuous and worse than at any previous time. This period coincided with the birth of a baby, its daily bathing, washing of diapers and complete care of the home.

A characteristic eruption involved both palms and several fingers of each hand. Repeated direct microscopic examinations and cultural studies of the roofs of the vesicles and the scales by one of us (J. H. S.) did not reveal fungus.

Treatment consisted of use of potassium permanganate soaks in dilutions of 1:5,000, weekly exposures to ultraviolet rays and the application of boric acid ointment. Aluminum chloride in strengths of 10 to 20 per cent was used to curb a moderate hyperhidrosis of the palms that became evident as the dermatitis subsided. Under this regimen the intensity of the vesicular outbreaks became progressively less until apparent recovery took place in the spring of 1944.

The recurring vesicular outbreaks appeared with considerable regularity, as the following dates show: September 4, October 1 and 29, November 30, January 3 and 29, February 28 and March 27. The last attack, which consisted of not more than ten vesicles, followed a week of spring cleaning with considerable washing.

Case 2.—Mr. B., a 30 year old meat order clerk, was first seen on Aug. 28, 1942. He worked in a packing plant and handled raw, damp meat all day in a refrigerator room with the temperature in the fifties. He washed his hands frequently with soap powder and cold water. A characteristic eruption was confined to the ring and middle fingers and the forefinger of the right hand throughout the ensuing year. The original outbreak, which had started two months previously and was accompanied by secondary infection, responded to treatment sufficiently for the patient to return to his work after one month. During the next four months the dermatitis waxed and waned, and he treated the outbreaks himself.

In January 1943, the patient gave up handling meat and got a job as an elevator man, using gloves and sparing his right hand as much as possible. Treatment was renewed on Feb. 11, 1943, and after the obscuring dermatitis was quieted, definite vesicular outbreaks appeared on the following dates: March 20, April 26, May 30, June 19 and July 10. He was discharged on August 16 with the skin quite well and with no outbreak during the first part of August. The treatment was essentially the same as that used in case 1.

Examinations (by J. H. S.) did not reveal fungi, and biopsy of a lesion taken from the ring finger showed hyperkeratosis, parakeratosis and some edema of the prickle cell layer. The capillaries of the papillary layer were dilated and surrounded by an inflammatory infiltration, chiefly round cells. A similar infiltration was present about some of the sweat ducts.

COMMENT

This clinical entity has been given many names, such as dermatophytosis, id, bacterial infection, eczema due to food idiosyncrasies and occupational dermatitis. However, the fact that mycologic studies repeatedly give negative results, both by direct microscopic examination and by culture, does not substantiate the diagnosis of fungous infection. The usual absence of an active focus of fungous infection militates against the possibility of an "id." Elimination of suspected foods has produced no beneficial results. Absence from work or a change in occupation does not prevent relapses.

What is this disease? The answer at present can only be speculative. We believe that it is due to an endogenous rather than an exogenous factor, which is set to work when there is lowered resistance of the tissue caused by a severe dermatitis or by continuous or repeated irritation. This endogenous factor may be a toxin, circulating in the blood, set free by severe or continuous damage to the skin. This substance may act on the capillaries or sweat ducts in that area, causing a disturbance in their function and inflammation. The characteristic recurrence in cycles is contradictory to the theory of direct bacterial invasion of a tissue that has developed a lowered resistance as a result of a preceding

dermatitis. It is not, however, contradictory to a possible virus infection, since other infections caused by a virus, such as recurrent herpes simplex, may be recurrent and periodic.

Further histopathologic studies of the sweat ducts in the involved areas are being made. Attempts at reproducing the disease in healthy areas by inoculation with the contents of the vesicles are also being carried out, and the results will be included in a later report.

The strongest competitor of this recurrent pearly dermatitis is the currently ignored disease of pompholyx, whose clinical characteristics are closely parallel, but the etiologic sources of the two diseases are completely different. Knowles, Corson and Decker [1] say: "It is probable that pompholyx is due to absorption of toxins from diseased areas, among them being dermatophytosis. The tonsils are very often responsible, as are teeth, sinuses, appendix and other septic foci. This would mean hematogenous transmission." Ormsby and Montgomery [2] say: "It [pompholyx] is said to occur in persons of nervous temperament and in those who are in a depressed mental state ascribed to worry and overwork. It is considered by most observers to be a neurosis, by some to be of toxic origin, the toxin being produced in the gastrointestinal canal."

Nowhere is it suggested that pompholyx may be a sequela of chronic irritation sustained in either industrial or private life. On the other hand, in a carefully appraised case, White [3] specifically rules out pompholyx as having any causal relation to industrial dermatitis. He writes as follows:

> The diagnosis was given of "trade eczema" in a man working at a "jig" in which he was using a series of different substantive dyes. As the symptoms lasted many months further enquiry elicited the following facts. The patient was eventually discovered to have a typical pompholyx (Tilbury Fox's "dysidrosis") unsuspected and therefore untreated by the medical attendant who gave the certificate. The rash continued to erupt periodically months after the man left his work. Before the man left work the author explained to him that, working or playing, the rash was liable to recur. During a two and one-half months' holiday at home and after he returned to a dry and wholesome job, the eruption returned at intervals on his fingers. This is the usual custom or habit of cheiropompholyx, a not very common complaint. In this case the breaking out was independent of . . . his work. It was the result of an internal toxin or irritant at present unexplainable, causing inflammation on these parts. After the cuticle has become broken by disease, it would have been aggravated or made worse by his first occupation. Aggravation does not last indefinitely.

Lane,[4] in his discussion of the papers by White, Weidman and Williams read before the American Dermatological Association at the Golden Anniversary Meeting in Philadelphia, made the following remarks:

> I believe that in the cases of epidermophytosis due to injury two classes will be found. In the first group are cases in which the industrial factor is the prime consideration. In the last year I saw a man who was a wool puller in one of the shops in Boston, who had, I believe, a fungous infection due directly to his work.
>
> There is another group in which the infection occurs as a complication of a preexisting injury or dermatitis of industrial origin, but these infections will be classed as due to injury, and for them compensation can be claimed. I have seen a case of this type in a treer in a shoe shop who had a definite dermatitis associated with his work. The condition cleared up almost completely and then intradermal vesicles developed on the lateral surfaces of the fingers with fairly sharply outlined patches on the hands. I was not able to obtain the fungus in the case, as I was in the first, but still I reported it as a definite complication of industrial disease. I believe this is by far the largest of the two groups that I have mentioned for I have seen several such cases.

In our opinion this recurrent pearly dermatitis is a causally related complication of a previous chronic irritation of the skin originating in industrial or private life. Current dermatologic opinion unanimously agrees that pompholyx is a constitutional disorder which has no relation to a previous dermatitis irrespective of cause.

Testimony at this time that pompholyx is compensable as a complication of industrial irritation of the skin would subject the dermatologist to the charge that his opinion is contrary to prevailing dermatologic thought. Under these conditions, it is our opinion that the acceptance of a new clinical entity of this recurrent pearly dermatitis by the name of "dermatitis margaritata recurrens" is justified.

ABSTRACT OF DISCUSSION

Dr. John G. Downing, Boston: I wish to congratulate Drs. Blaisdell and Swartz on their excellent presentation. All those in industrial dermatologic practice have noted this type of recurrent eruption, and one is at a loss to explain to the insurer why an eruption which one stated would be well in six weeks persists for many months. However, I am opposed to introducing this type of recurrence of an old dermatitis as a new entity, for it will involve many complications at hearings at the Industrial Accident Board. When a dermatitis persists for over three months, my first thought is: " Did I make a correct diagnosis?" My second thought is: "Is this a case of pompholyx—cheiropompholyx—dyshidrosis, or one of the other well known

1. Knowles, F. C.; Corson, E. F., and Decker, H. B.: Diseases of the Skin, Philadelphia, Lea & Febiger, 1942, p. 189.

2. Ormsby, O. S., and Montgomery, H.: Diseases of the Skin, Philadelphia, Lea & Febiger, 1943, p. 368.

3. White, R. P.: Occupational Affections of the Skin, New York, Paul B. Hoeber, 1928, p. 78.

4. Lane, C. G., in discussion on papers of White, Weidman and Williams, Arch. Dermat. & Syph. **15**:467 (April) 1927.

vesicular eruptions of the hands so well described by Fred Wise?" In my experience these lesions have also appeared on the inner flexor aspect of the forearms.

Dr. Samuel W. Becker, Chicago: This excellent paper is certainly timely. Dr. Fred Wise gave a paper on this subject in Philadelphia, and I discussed the problem at the American Academy of Dermatology and Syphilology in 1940 and at the Michigan State Medical Society in 1941.

Some years ago I went through the literature, trying to convince myself as to just what this condition might be. In 1873 Tilbury Fox wrote a paper which is similar to the one by Drs. Blaisdell and Swartz.

The patients are of a nervous type and present hyperhidrosis. Tilbury Fox reasoned, we now know wrongly, that the vesicles resulted from a damming up of the sweat ducts. It is now believed that the hyperhidrosis and the eruption are both on a nervous basis. He called the disease "dyshidrosis." In the official nomenclature, it is now called pompholyx.

As I have studied these patients, I believe that this eruption on the fingers, the vesicular eruptions of the palms and the vesiculopustular eruptions which have been called recalcitrant eruptions of the palms and soles all fall in the functional group. In other words, at least at the beginning there is no influence from the outside which has produced the eruptions. The cause is purely internal.

As I have studied the patients, rather than the eruption, I find that they are the type of persons who are entitled to develop functional disease. These patients never show positive reactions to patch tests, which further discounts the idea of external irritation as causative.

However, to make the matter more difficult, these patients do sometimes have positive reactions to tests to metals. I had 2 patients within the last few weeks, 1 of whom was reported to have reacted positively to nickel and 1 to zinc chromate material used on airplane wings. I repeated these tests, and obtained a reaction but not primarily vesicular as seen in epidermal hypersensitiveness. It was primarily erythema plus tremendous edema, and following edema there was secondary vesiculation. So, I do not consider this a positive reaction to a patch test. These patients will also show positive dermal reactions either by the intradermal or by the scratch technic.

I am much more optimistic about treatment than Drs. Blaisdell and Swartz. I place these patients on a combination of dermatologic management and treatment for functional disease. I use potassium permanganate wet dressings, calamine liniment and 3 per cent ichthammol ointment. After about a week, crude coal ointment is added. I think 5 per cent crude coal ointment aids more than anything else in making the keratinization of the skin disappear. The patients are advised to take a daily nap. I advise them to buy a sunshine lamp. As a result, many of these patients have been relieved, not always completely. They have occasional recurrences, because, if one assumes that a functional disease represents a perversion of the sense of fatigue, when the ordinary individual would get tired these people have outbreaks of blisters. If, however, they cooperate reasonably well, they get acceptable results.

Dr. Samuel M. Peck, Bethesda, Md.: Eruptions on the hands due to fungi are infrequently met with in industry and they can be said to play a relatively unimportant role as the cause of lost time in industry. Unfortunately the diagnosis of dermatophytoses and dermatophytids of the hands is made too frequently without adequate clinical and laboratory findings to justify this diagnosis.

It has been possible to show that the fact that a fungous infection is present which leads to an allergy to the fungi and their products is no reason for assuming that the affected person is predisposed to other allergic diseases. Fungous infections of the hands and feet play a role as a predisposing factor only by allowing more intimate contact between the living cells and the primary irritant and sensitizers. The same sort of a role is played by cuts and abrasions in the skin. The dermatologist who has not had the opportunity of studying large numbers of workers has a wrong impression of the relative importance of dermatophytosis as a causative factor in loss of time and industry. He is led to such an impression because chronic or recurrent cases with eruptions on the hands and feet accumulate in his office so that in his own clinical material he may see a high incidence of such eruptions differentiated from fungous infections or due to fungi.

Before the authors can be sure that they have a new clinical entity in the syndrome which they are describing, they must prove that the eruption in their cases is neither an epidermatophytid nor an ordinary contact dermatitis. This in my opinion they have failed to do because neither a trichophytin test nor patch tests have been made in most instances.

Dr. Joseph Grindon Sr., St. Louis: We all are familiar with these cases in which a mycosis can be excluded, and are apt to think that they are due to contact conditions, and probably many of them are due to nickel or whatever it may be, or something in the industries, but in some cases they are not of that sort. I do not care whether they are called pompholyx or given some other name.

I think it was the late Dr. Robinson of New York who, a good many years ago, showed that these vesicles were not due to dyshidrosis, not connected with the sweat glands.

There is one type of this disease which is rare, but I think you have all occasionally encountered it; that is an eruption affecting one finger of one hand, which has recurred for many years. About forty years ago Blaschko of Vienna, I think, called it herpes digitalis, which is a good name for it. In those cases and in other cases of vesicular eruption of the palms and fingers in which one can assign no probable cause I use a method which has been successful in a number of cases. I use neither local application nor the roentgen rays nor the ultraviolet ray, but (please don't laugh at me) I give the patients 1/30 grain (0.001 Gm.) of strychnine three times a day. Sometimes there is rapid recovery. If there have been recurrences, they have not come to my knowledge.

Dr. Samuel Ayres Jr., Los Angeles: One brief word to corroborate Dr. Blaisdell's findings in these cases in which the eruption continues to recur after the patient apparently is well. It undoubtedly is a nonspecific sensitivity that has persisted, or I think it is following the original contact irritation.

I do not think that Dr. Becker's statement that a reaction to a test with a piece of nickel or zinc chromate merely because it shows redness or edema and no vesicles is not considered positive should go without question. I think it is too important a matter in the question of industrial dermatologic practice. The insurance companies would like to have the dermatologist say that the reaction to a patch test was negative if vesicles did not appear. I think, for the record, that point ought to be made.

Dr. Jacob Swartz, Boston: Both Dr. Blaisdell and myself wish to thank those who have discussed this

paper. We expected a good many more hot blows; I guess we got off lightly. We realize the risk we took in describing an entity, but this particular disease we do see in Boston, and I was happy to hear that you see it in other places. It is a typical clinical condition, as I will mention in a few minutes. In our cases, we did not see the eruption on the inner aspect of the elbows or forearm as Dr. Downing mentioned.

The functional aspect Dr. Becker stated. I am particularly interested in functional dermatosis. Although I have not written on the subject, I have studied such cases carefully. In these particular patients we have not noted, at least emotionally and otherwise, the findings that we noted in the cases of atopic dermatitis or neurotic excoriations. Cheiropompholyx resembled our described entity the closest. But, as already stated in our paper, such authorities as Ormsby, Knowles, Prosser, White and others specifically state cheiropompholyx is not causally related to industry or chronic irritation but is the result of a constitutional disease to which it is related.

Wise and Wolf in the *New York State Journal of Medicine* in November 1940, discussing the diagnosis and treatment of recurrent vesicular eruptions of the hands, state: "In cheiropompholyx there is no history of contacts." In this entity there is a history of a preceding industrial dermatitis or, as in the case of the housewife, a continuous exposure to irritants or sensitizing agents.

Against the argument that this entity is a simple contact dermatitis are the following facts:

1. Absence of lesions on the dorsum of the hands and other areas particularly involved in the case of contact dermatitis.

2. Absence of preeruptive erythema.

3. Presence of deep-seated, pearly vesicles.

4. Absence of eczematization subsequent to regression of lesions.

5. Periodicity of relapses and remissions after the avoidance of the original contacts which brought on the attack of acute dermatitis that preceded this clinical picture. The periodicity is characteristic in these cases.

This entity is not an "id," because there is no clinical evidence of an active fungous infection on the feet and elsewhere on the skin, and we looked for it closely, unless one argues a discolored fifth toe nail, which most likely harbors the organism, is the focus for the id. Such a possibility is most unlikely.

Against the theory that these pearly vesicles are the result of bacterial invasion on previously injured skin are:

1. Absence of clinical inflammation such as erythema, pustulation and crusting.

2. In the few cases tried, negative results obtained in an attempt to reproduce such lesions by inoculating into the skin in adjacent areas the contents of the pearly vesicles.

We realize it is rather confusing to start a new name and new entity, but is it not more confusing to have Dr. X talk about this disease under a certain name or type and Dr. Y under other names, as we have seen here, and not understand each other? Is it not better to call it one name so we can understand each other? Then, perhaps, the causation will be found faster.

NEVUS SEBORRHEICUS ET SUDORIFERUS

A UNILATERAL LINEAR PHYSIOLOGIC ANOMALY

HARRY L. ARNOLD JR., M.D.
HONOLULU, TERRITORY OF HAWAII

Simple hyperplasia-hypertrophy and hypersecretion of the sebaceous and sweat glands in a circumscribed area of skin do not appear to have been mentioned in any published account of nevi and adenomas of the sebaceous or sweat glands.

Woolhandler and Becker [1] have clearly divided the sebaceous gland overgrowths into the following categories:

Adenoma sebaceum (Balzer)
Adenoma sebaceum (Pringle)
Acquired (senile) adenoma sebaceum
Keratotic adenoma sebaceum
Sebaceous gland adenoma
Nevus sebaceus
Nevus pigmentosus et sebaceus

Whatever this nomenclature may leave to be desired, the categories themselves are reasonably clearcut, and the case to be reported here does not correspond to any of them. These authors did suggest that hyperplasia of sweat glands may on rare occasions be associated with nevus sebaceus; but in that lesion the sebaceous glands are so large and numerous as to form yellowish papules or masses, and the basal layer of the sebaceous acini is found to be absent in sections studied histologically. Becker and Obermayer [2] naturally (and more recently) concurred in this view and amplified it somewhat.

Wolters [3] confirmed the statement about the occasional association of hyperplasia of sweat glands with nevus sebaceus; his patient, however, did not appear to have shown evidence of active hypersecretion of either sweat or sebum.

Way [4] and Warren and Warvi [5] did not refer to any lesion of the sebaceous glands characterized by hyperplasia and hypersecretion, much less by concomitant hypersecretion of sweat.

Linear unilateral nevi consisting principally of sebaceous glands or of comedos have been reported many times. Sweitzer and Winer [6] have summarized the literature (some 30 cases) on nevus unilateralis comedonicus, which typically has a linear and unilateral pattern but consists solely of dry comedos with or without surrounding inflammation and hypoplastic or even absent sebaceous glands. Cohen [7] has reported such a case under the name nevus acneiformis unilateralis. The sweat glands are apparently unaffected in this disease. Poor [8] was cited by Stelwagon [9] as having concluded that there

Fig. 1.—Linear configuration of the lesion, hypopigmentation, comedos and the huge droplets of sweat limited to the involved area.

1. Woolhandler, H. W., and Becker, S. W.: Adenoma of Sebaceous Glands (Adenoma Sebaceum), Arch. Dermat. & Syph. **45**:734 (April) 1942.

2. Becker, S. W., and Obermayer, M.: Modern Dermatology and Syphilology, Philadelphia, J. B. Lippincott Company, 1940, p. 579.

3. Wolters, G.: Ueber ein Fall von Naevus epitheliomatosus sebaceus capitis, Arch. f. Dermat. u. Syph. **101**:197, 1910.

4. Way, S.: The Sebaceous Glands: Their Histopathology and Role in Diseases of the Skin, Arch. Dermat. & Syph. **24**:353 (Sept.) 1931.

5. Warren, S., and Warvi, W. N.: Tumors of Sebaceous Glands, Am. J. Path. **19**:440 (May) 1943.

6. Sweitzer, S. E., and Winer, L. H.: Nevus Unilateralis Comedonicus: Nevus Follicularis Keratosus of White, Arch. Dermat. & Syph. **26**:694 (Oct.) 1932.

7. Cohen, E. L.: Nevus Acneiformis Unilateralis, Brit. J. Dermat. **55**:297 (Dec.) 1943.

8. Poor, cited by Stelwagon.[9]

9. Stelwagon, H. W.: A Treatise on Diseases of the Skin, Philadelphia, W. B. Saunders Company, 1910, p. 639.

is a type of nevus sebaceus, which he called nevus sebaceus circumscriptus asymmetricus, which is usually linear. It does not appear, however, that this lesion is associated with abnormality of the sweat glands. It was probably an example of the same type that was reported by Maloney [10] as nevus sebaceus linearis, which he described as consisting of yellowish closely set papules, some topped by comedos, histologically indistinguishable from adenoma sebaceum.

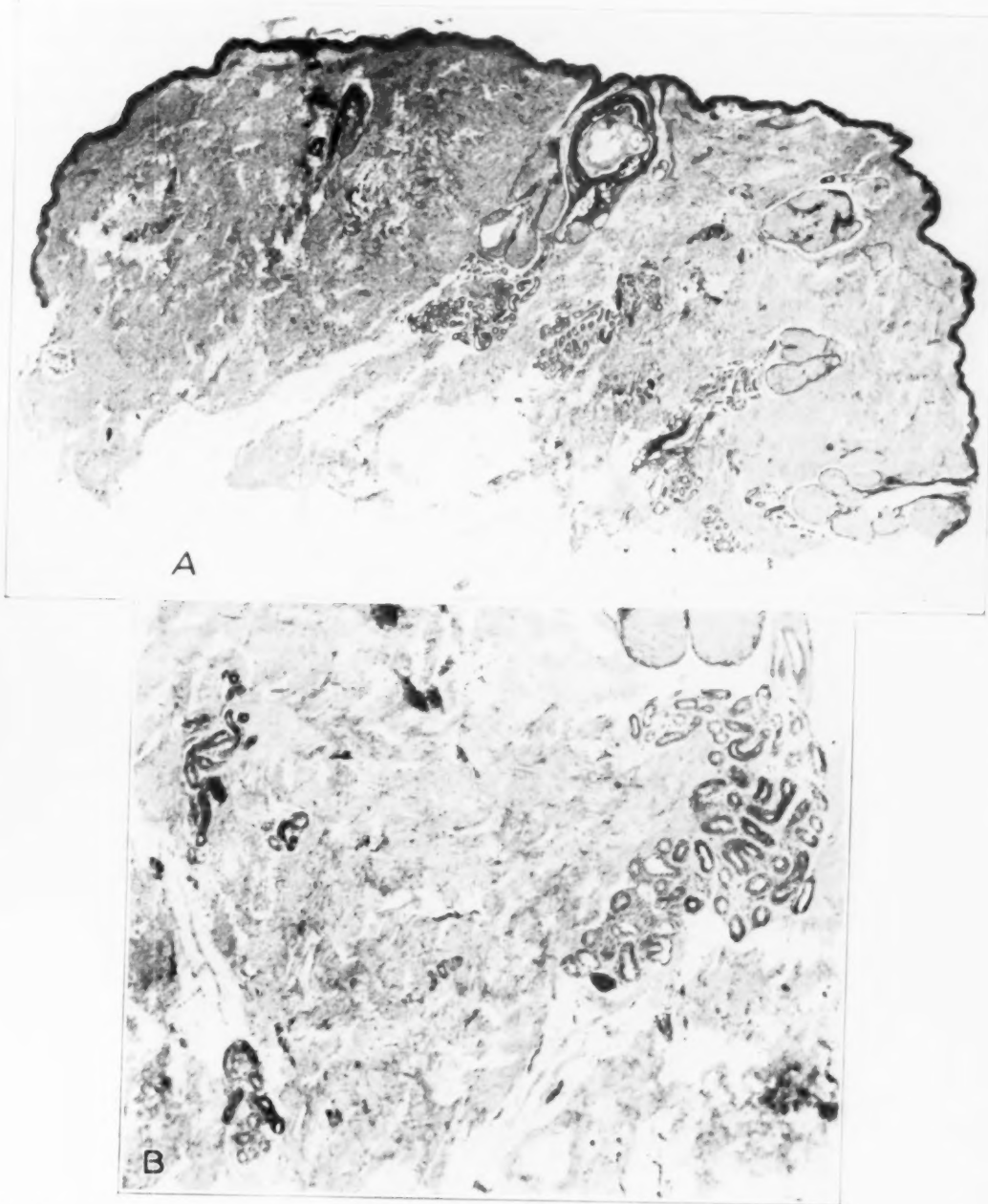

Fig. 2.—*A*, from left to right, three small pilosebaceous and sweat gland elements outside the lesion and four consecutive, greatly enlarged pilosebaceous and sweat gland elements, one showing an obliquely cut comedo, in the involved area. Note the numerical increase of the coils of the sweat gland. Hematoxylin and eosin; × 6. *B*, higher magnification of the transition from normal to abnormal glands. Note the striking difference in size of the sweat gland acini. Hematoxylin and eosin; × 20.

As to the reported association of abnormality of the sebaceous glands with hypersecretion or hypertrophy of the sweat glands, no such lesion

10. Maloney, E. R.: Nevus Sebaceus Linearis (Case Presentation), Arch. Dermat. & Syph. **30**:167 (July) 1934.

was mentioned by Gates, by Warren and Warvi [11] or in Suttons' [12] or Becker's [2] texts.

REPORT OF A CASE

T. S., a 20 year old Japanese man, was first seen in December 1943 because of a lesion on the left side of his neck which had been present for nearly three years. It had caused no itching or pain but was periodically annoying to him because of its extreme moistness and oiliness and because of the blackheads that frequently developed in it.

Examination disclosed a healthy, athletic-looking young Japanese man whose skin was clean and free from significant lesions; he had in particular no acne or dandruff. On the left side of his neck, about 5 cm. below and posterior to the ear, there was a sharply outlined, irregularly oval hypopigmented macule about 3 by 4 cm., which extended forward and upward along a path about 1 cm. broad to a point just anterior to the tragus of the ear. This macule was decidedly moister than the surrounding skin and was studded with numerous large soft greasy comedos. While the lesion was being photographed, it was observed under the heat of the lights to become beaded with huge droplets of sweat, far out of proportion to the amount of perspiration on the surrounding skin (fig. 1).

Biopsy was performed, and the sections showed that the pilosebaceous elements within the lesion were many times larger than those outside it and each had immediately underlying it one or two large coil gland acini (fig. 2). There were no other significant changes noted. Dr. Fred Weidman, to whom sections were sent, expressed the opinion that the coil glands appeared to be in the exhausted rather than in the resting stage.

COMMENT

The indolence of this lesion, its sharp localization, its unilateral and linear configuration and its lack of any apparent causative factor all support the view that it is a nevus, notwithstanding its having first appeared when the patient was 17 or 18 years old. There is nothing about the histologic changes to contradict this view, and indeed they completely fail to support any other; certainly they show no evidence of neoplasia. The degree of hypertrophy or hyperplasia of the glands is far from sufficient to warrant classifying the lesion as an adenoma.

As nearly as can be judged, the sebaceous and sweat glands appear to share about equally in the process; neither seems to be enlarged out of proportion to the other.

Finally, the most striking aberration from the normal is in the function of these glands rather than in their structure; the profuse outpouring of sweat under the influence of external heat (or physical exertion) is remarkable, though no more so than the continued outpouring of sebum evidenced by the greasiness of the lesion and by the large soft greasy comedos occupying almost every hair follicle.

SUMMARY AND CONCLUSIONS

A linear unilateral lesion in which the sweat and sebaceous glands were larger and much more actively secreting than those in the surrounding skin was observed.

This lesion seems to represent an aberration of function rather than of structure; it appears to be a physiologic secretory nevus involving the sebaceous and eccrine glands of the skin.

Dr. I. L. Tilden, of the Department of Pathology of The Clinic, Honolulu, made the photograph, prepared the histologic sections, and made the photomicrographs.

The Clinic, 881 South Hotel Street.

11. Gates, O.; Warren, S., and Warvi, W. N.: Tumors of Sweat Glands, Am. J. Path. **19**:591 (July) 1943.

12. Sutton, R. L., and Sutton, R. L., Jr.: Diseases of the Skin, ed. 10, St. Louis, C. V. Mosby Company, 1939.

DEMENTIA PARALYTICA AND WASSERMANN REACTION OF THE SPINAL FLUID

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CLARINDA, IOWA

It is the consensus of physicians treating dementia paralytica that an indispensable factor in support of the diagnosis is a positive Wassermann reaction of the spinal fluid. The literature dealing with the serologic reaction reflects the same idea. Wechsler [1] stated that "the blood and, especially, spinal fluid Wassermann are positive in practically 100% of cases." According to Stokes,[2] "The negative spinal fluid is better presumptive evidence of the absence of syphilis in the neurological field than is the negative blood Wassermann reaction in general medicine." He concluded that "a negative spinal fluid, even in the presence of a neurological condition, almost excludes general paresis from the diagnosis." Stokes continued: "The question of the existence of seronegative paresis is as yet undecided, but there are some indications that general paresis in a preponderately vascular phase may be almost, if not quite, seronegative in the spinal fluid, at least after treatment, and still be active and symptomatically progressive. Cases of this sort, however, are so rare that they need hardly enter into the diagnostic rules applicable to ordinary practice." Becker and Obermayer [3] postulated unequivocally that "in typical paresis the test is always strongly positive throughout all concentrations." Grinker [4] confirmed this and elaborated that statement by adding that "if only 0.22 cc. of fluid is required to give a positive reaction, there is great probability that the patient is suffering from either paresis or taboparesis; it is much less probable that he is the subject of other neurosyphilis or beginning tabes. In many more cases of neurosyphilis and tabes the Wassermann reaction becomes positive by the use of larger quantities of fluid—from 0.4 to 1 cc." But he pointed out "that in general paresis the cerebrospinal fluid gives a positive reaction with the smallest quantity—0.05 to 0.2 cc.—a possible point of differentiation from other types of neurosyphilis. An additional point is the fact that not only is the positive Wassermann reaction seen in its greatest intensity in general paresis, but once present it is with difficulty changed into a negative one and often becomes Wassermann fast. . . . In our opinion whenever a case clinically diagnosed as general paresis presents negative findings in blood and cerebrospinal fluid, the diagnosis of paresis should be reconsidered until such time as further examination shall either prove or disprove the clinical diagnosis. . . ." These quotations could easily be augmented and substantiated by statements of many other authors in the international literature as well as by the conclusions of any practitioner dealing with dementia paralytica.

These rules seem acceptable only with certain reservations, however. In the following series of cases deviations from the aforementioned observations became evident. The patients were seen exclusively at the Clarinda State Hospital within a few months previous to the writing of this report. In the histories all details which have no bearing on the Wassermann reactions of the spinal fluids are omitted. All serologic procedures were performed at the Iowa State Department of Health, Iowa City, the vast experience of whose staff guarantees accurate results. Care was taken that antisyphilitic treatment which might have influenced our tests was not given at the time.

Case 1.—A. B. C. was treated for eight months in 1939 at the University Hospitals, Department of Neurology, State University of Iowa. At that time encephalograms showed slight cortical atrophy. He had an intelligence quotient of 84 (dull normal). The diagnoses were neurosyphilis and dementia paralytica. The case was rather unusual in the absence of a positive Wassermann reaction of the spinal fluid, but the positive colloidal gold curve, the general history and other evidence all bore out the diagnosis. There was no evidence of gumma or tumor. In December 1939, in Clarinda State Hospital, his neurologic status, including the pupillary reactions, was found to be still normal, but he exhibited epileptiform seizures, showed destructive and suicidal

From the Clarinda State Hospital.

1. Wechsler, I. S.: Textbook of Clinical Neurology, ed. 4, Philadelphia, W. B. Saunders Company, 1939, p. 469.

2. Stokes, J. H.: Modern Syphilology, Philadelphia, W. B. Saunders Company, 1939, p. 161.

3. Becker, S. W., and Obermayer, M. E.: Modern Dermatology and Syphilology, Philadelphia, J. B. Lippincott Company, 1940, p. 750.

4. Grinker, J.: Syphilis of the Nervous System, in Tice, F.: Practice of Medicine, Hagerstown, Md., W. F. Prior Company, Inc., 1935, vol. 9, chap. 8, p. 799.

tendencies and became self assertive. The patient had no insight concerning his mental condition, but he showed remnants of intelligence. In the following year the seizures increased, and he became confused and irritable. In 1941 he received malaria therapy and improved considerably. His seizures ceased with adequate treatment and did not reappear when use of the drugs was discontinued. His mental condition grew so favorable that he could be paroled; he was and is able to make a living by holding a job. During 1943 the Wassermann reaction of the blood became positive again after having been negative repeatedly in 1941 and 1942. Chemotherapy was started once more. The neurologic findings remained negative, and he appeared in satisfactory mental condition throughout this time. Results of the tests of the spinal fluid were:[5]

	Wassermann Reaction	Colloidal Curve	Cells	Globulin
4/15/39	Negative	5555400000	9	Negative
11/25/39	Negative	0000000000	2	Negative
9/22/42	Negative	0000	8	Negative
3/ 8/43	Negative	11000	2	Negative

The patient is now in excellent physical condition.

Case 2.—G. C. was admitted to the hospital in June 1942. His pupils were unequal and reacted sluggishly to light and in accommodation; the Romberg test elicited a moderate response; the right patellar reflex was present, but the left one was missing. The patient had auditory hallucinations; he was forgetful, especially in regard to recent events. Besides, he was delusional and confused, and his orientation and intelligence were poor. At times he was violent. His syphilis had been acquired nineteen years before. The antisyphilitic chemotherapy he had received had been insufficient. The serologic tests of the blood at the time of his admission, October 20, showed positive Kolmer, Kahn and Kline reactions. In tests of the spinal fluid, the Wassermann reaction was found to be negative; faint traces of globulin were observed; 8 cells were counted and the colloidal mastic curve was 55430. He was treated with a course of oxophenarsine hydrochloride, tryparsamide, thio-bismol and potassium iodide till February 1943. He calmed down greatly, but speech was slurred and he showed progressing mental deterioration. The serologic reaction of the blood in February 1943 was again positive in the Kolmer, Kahn and Kline tests. The Wassermann reaction of the spinal fluid was now positive; there were 8 cells and faint traces of globulin, and the colloidal mastic curve was 55430. The patient was inoculated with malaria at that time, but he died one month later. The physical and mental observations, the results of laboratory investigations of blood and spinal fluid and the subsequent course of the disease prove the diagnosis of dementia paralytica beyond doubt. It was also proved by the positive Wassermann reaction of the spinal fluid in February 1943, but on his admission the same reaction had been negative.

Case 3.—H. B. was admitted to the hospital in October 1940. He had had syphilis for four years, and several courses of antisyphilitic chemotherapy in 1940 had produced negative Wassermann reactions of the blood and spinal fluid. In the spring of 1942 the Wassermann reaction of the blood became positive again. Mental changes were observed in the same year, such as forgetfulness, grandiose ideas about money, recklessness in driving and evidence of a fighting spirit. These symptoms turned to worry about financial affairs. He had a feeling of inferiority and a tendency to isolate himself. Furthermore, he suffered from insomnia. The physical observations at the time of his admission disclosed the following picture: The pupils were normal; the Romberg sign was not elicited, and the patellar reflexes were present. The reactions of the blood in the Kolmer and Kline tests were negative. On Oct. 15, 1942 the Wassermann reaction of the spinal fluid was doubtful; five days later, on October 20, however, it was negative, although no therapy of any kind had been given between these dates. Besides, the spinal fluid showed 14 cells; the globulin reaction was positive for dementia paralytica, and the colloidal mastic curve was 55432. The diagnosis was based on the unmistakable mental picture and on the reactions of the spinal fluid. The further course of the disease also favored the diagnosis. After preliminary treatment with tryparsamide, mapharsen and thio-bismol, malaria treatment was begun, and after thirteen chills the same chemotherapy as before was administered. The patient improved so much that in April 1943 he could be discharged in care of the family physician as a normally reacting person.

Case 4.—A. B. was admitted to the hospital in September 1939 with the diagnosis of dementia paralytica. Mentally he had shown forgetfulness, childish behavior, irritability and the habit of talking to himself. He was always irrational, optimistic and joyous. This condition did not change. The physical signs were: unequal but round pupils which reacted not at all to light and only sluggishly in accommodation, slight intentional tremor but no ataxia and good coordination. The following serologic reactions were observed: The reaction of the blood was positive in the Kolmer and negative in the Kline test; the Wassermann reaction in the spinal fluid was negative. Six months later the reaction of the blood was doubtful in the Kolmer test but was negative in the Kahn and Kline tests. The mental picture has not changed up to the time this report was written. The diagnosis was based on the patient's mentality and on the slightly abnormal pupillary reactions, but it is not absolutely beyond doubt and is open to criticism. In December 1942 the spinal fluid showed 8 cells; the globulin reaction was negative for dementia paralytica, and the colloidal mastic curve was 32100. Results of these tests were favorable, although no malaria treatment or chemotherapy had been applied.

Case 5.—A. L. was admitted to the hospital in January 1943. Her mental disturbances had begun a year previously with abnormal worrying about financial matters; she was noisy and talkative and frequently sang. At the same time the patient became aggressive; often she had crying spells. Later she was quiet and isolated herself. She repeatedly uttered hypochondriac complaints of various kinds. Her speech was slurred at times. The only abnormal physical signs were a rather poor coordination and high knee reflexes. In February 1943 there were positive reactions of the blood in Kolmer, Kahn and Kline tests. In the spinal fluid, the Kolmer reaction was negative; there were 10 cells and no excess globulin; the colloid mastic curve was 43210. With antisyphilitic chemotherapy, which preceded and followed the treatment with malaria, the patient improved. She is still under an antisyphilitic regimen. The serologic reaction of the blood remains positive. Close daily observation of her mental condition corroborates the diagnosis of dementia paralytica, although it is somewhat questionable when all physical and serologic evidence is considered.

Case 6.—B. B. was a "doorstep" child, having been found in 1919, with signs of prenatal syphilis, gonorrhea and otitis media. Her upper incisors were

5. These tests were made at the Clarinda State Hospital, where since 1942 the colloidal mastic curve has been used instead of the colloidal gold curve.

notched. The Wassermann reaction of the blood was positive in 1921 when tested on at least seven different occasions; it became negative after intense antisyphilitic chemotherapy with neoarsphenamine and with preparations of mercury and of iodine. It became positive again and remained so in spite of thorough treatment and malarial therapy in September and October 1942. Therefore more chemotherapy was given. In July 1937 the spinal fluid had been examined at the Iowa City University Hospitals and was found normal in every respect. In June 1942 the right pupil was slightly irregular, but there was normal reaction on both sides; the fingers showed poor coordination. The spinal fluid again was normal in June 1942; the reactions of the blood in Kolmer, Kahn and Kline tests were all positive. The mental picture showed severe changes. The patient, who had been reared in a law-abiding, well-to-do family and had enjoyed the best education, stole a car, sold it, borrowed $530 under false pretense and became egocentric, quarrelsome and absolutely amoral. She had grandiose ideas, was forgetful and irritable and had outbursts of temper. She called herself oversexed and also showed homosexual tendencies. During treatment she improved considerably and could be paroled in 1943 in care of her foster parents and the family physician.

CASE 7.—C. O. was admitted to the hospital in January 1943 with the diagnosis of dementia paralytica. There were signs of involvement of the pyramidal tract; there was a fine tremor and poor coordination of the extremities. The Romberg sign and the Babinski reflex were elicited, as was the foot clonus. The patient was forgetful; he had loss of memory and of initiative. His intellect and judgment were impaired, and he was disoriented as to time and place. The reactions of the blood in the Kolmer, Kahn and Kline tests were positive and remained so even after intense antisyphilitic chemotherapy was applied and after he had been inoculated with malaria. But before any treatment had been started, the spinal fluid showed a negative Wassermann reaction, 6 cells and a colloid mastic curve of 55554. The patient, who is still under treatment, has improved considerably but is not yet normal.

COMMENT

The cases mentioned were observed during a relatively short period. It is possible that one could find an even larger series at this hospital alone, were it not for the fact that not all registered patients can be reexamined frequently, for reasons common to all institutions in these days. As stated before, a strict critic might find reason for debate concerning some of these cases. Much will depend on how far the mental picture can be considered as a deciding factor in the diagnosis of dementia paralytica. In a syphilitic patient an anorganic psychosis can always develop, either before or after his infection. But if one observes the signs and symptoms of dementia paralytica, it is rather difficult to refute a relationship of the syphilis with the psychotic features. Dementia paralytica may well be the cause in any given case, even though some of the fundamental signs of this disease are missing.

A thorough study of the literature previously cited reveals that some authors qualified their statements concerning the necessity of a positive Wassermann reaction of the spinal fluid by describing it as being only "almost" or "nearly" indispensable. In the first case reported here, the Iowa City University Hospitals established the diagnosis of dementia paralytica, emphasizing that the Wassermann reaction of the spinal fluid was negative.

I certainly do not wish to convey the impression that this report of a few cases, some of which may be questionable, is intended to overthrow a doctrine which has been fortified by hundreds and thousands of examples and which has led a man like C. O. Cheney [6] to describe patients with dementia paralytica as persons "showing rapidly or slowly progressive organic intellectual and emotional defects with physical signs and symptoms of parenchymatous syphilis of the nervous system and completely positive serology including the positive gold curve." In order to modify such well based conclusions, much larger series of cases would have to be examined and numerous statistics would have to be compiled. But the deviations from the usual are worth recording and present a stimulus for further investigation. I believe that the majority of persons with dementia paralytica in the United States are admitted to state institutions. If the patients treated in some of these hospitals should be checked from this serologic point of view, the purpose of this paper would be accomplished.

SUMMARY

It is suggested in the literature that dementia paralytica rarely occurs in association with a negative Wassermann reaction of the spinal fluid.

Seven cases were observed at the Clarinda State Hospital, Clarinda, Iowa, which may bear out the suspected possibility that dementia paralytica may coincide with a negative Wassermann reaction of the spinal fluid.

It is considered possible that a number of similar cases could be found and that it is of importance to study the problem further.

6. Cheney, C. O.: Annals of the National Committee for Mental Hygiene, ed. 10, New York, National Committee for Mental Hygiene, 1942.

MELANIN PRODUCTION IN SKIN

II. FURTHER HISTOCHEMICAL OBSERVATIONS

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An earlier report [1] dealt with the influence of protoplasmic poisons, such as potassium cyanide and sodium fluoride, and of alkalinity on the Meirowsky phenomenon (the increase in epidermal pigment in excised or cadaver skin when incubated at 37 C. and higher or on exposure to ultraviolet rays). These poisons served to accelerate the formation of pigment. It was further reported that in the experiment in which the solution containing the skin sections was overlaid with liquid petrolatum there was demonstrated an increased acceleration in pigment production. On the basis of this evidence it was suspected that atmospheric oxygen possibly was not required for the production of this phenomenon.

Earlier investigators of this phenomenon all came to the conclusion that atmospheric oxygen was required for its elicitation. The most recent and comprehensive studies of this problem, by Miescher and Minder [2] and, incidentally published before the presentation of our [1] first paper, confirmed the earlier conclusions. Despite the unanimity with which we are confronted, we are led by our studies to question the truth of the conclusions of the earlier investigators [3] and to affirm that the phenomenon of Meirowsky can be produced without the aid of atmospheric oxygen. Since most speculative thinking about human pigment and the method of its production centers about the available pertinent facts evolved experimentally, it is important that all these facts be probed thoroughly and, particularly, that a detailed record be made of the investigative procedures followed.

The several reported [1] significant procedures followed by us in producing the Meirowsky phenomenon are briefly summarized in the following paragraph:

Sections of frozen skin, hardened for less than twenty-four hours in solution of formaldehyde U. S. P. diluted 1 to 10, were placed in open beakers and incubated for three hours at 37 and 56 C. The sections were immersed in the following solutions, with and without an overlaying of the aqueous solution with liquid petrolatum: (1) 0.85 per cent sodium chloride, (2) 0.1 per cent potassium cyanide and (3) 0.1 per cent sodium fluoride. Further, all three solutions were variously buffered with phosphate to give p_H values of approximately 5, 6, 7, 8 and 9.

It should be noted that our original contribution to the studies of this phenomenon was the demonstration of the efficacy of protoplasmic poisons to enhance the degree of pigment production, and that the only similarity of our procedures to those of earlier investigators was the part of our reported practice in which we incubated the frozen sections in isotonic solution of sodium chloride.

In probing the report of the suppression of a reaction, the investigator must distinguish among the fact of suppression, the procedures used and the explanations offered for its accomplishment. And so, without denying any of the earlier reports of suppression of the Meirowsky phenomenon, we challenge the correctness of the explanation for the mechanism of its achievement. Incidentally, we can have no insight into the true explanation of the suppression, for we failed to prevent its production.

This research was made possible by a grant from the Dr. Simon Baruch Foundation.

From the Skin and Cancer Unit of the New York Post-Graduate Medical School and Hospital, Columbia University.

1. Sharlit, H., and others: Histochemical Observations on Melanin Production in the Skin, Arch. Dermat. & Syph. **45**:103 (Jan.) 1942.

2. Miescher, G., and Minder, H.: Untersuchungen über die durch langwelliges Ultraviolett hervorgerufene Pigmentdunkelung. Strahlentherapie **66**:6, 1939.

3. See Miescher and Minder [2] for bibliography.

On us developed the burden not alone of seeking to suppress the Meirowsky phenomenon by a procedure calculated to supply an oxygen-free atmosphere but of establishing acceptable proof that such an atmosphere, free of oxygen, had been present and operating throughout the experiment, in spite of our failure to suppress the phenomenon. We used a nitrogen atmosphere in which to carry out the incubation of the frozen sections and therefore accepted (1) the inhibition of the enzymatic conversion of para cresol to a melanin by potato tyrosinase and (2) the suppression of the auto-oxidation of a properly buffered solution of dopa to a melanin as proof of the effective presence of an oxygen-free atmosphere. Experiments confirmed Minder's experience [4] disclosing that exceedingly small traces of oxygen were difficult to exclude, since it took repeated variations of procedure to acquire a setup that completely prevented the spontaneous conversion of dopa to melanin. Incidentally, such stringent requirements were not needed to inhibit the production of paracresol melanin.

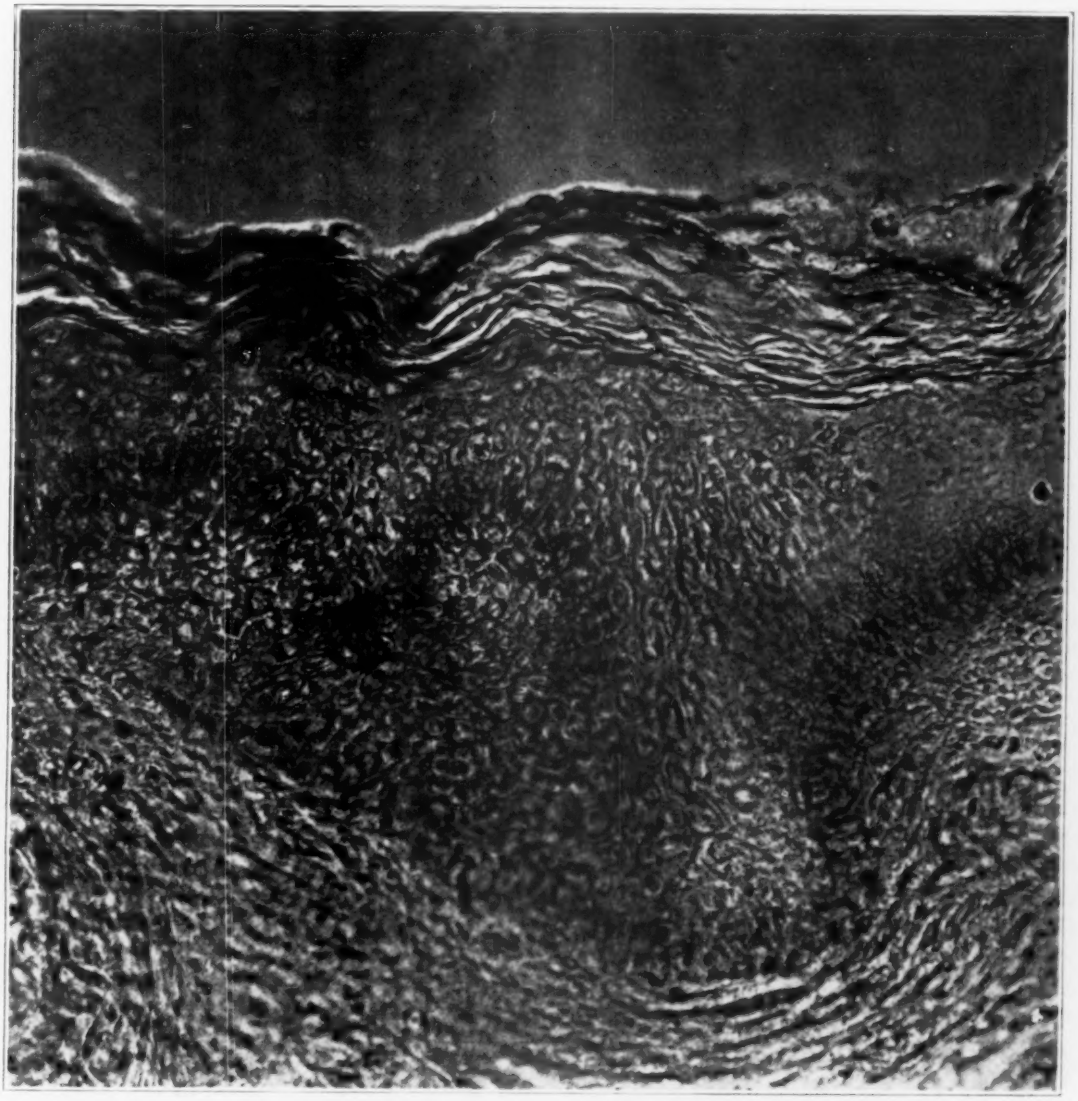

Fig. 1.—Control section, mounted immediately after cutting from the frozen block, discloses the amount and location of pigment originally present in the section.

4. Minder, H.: Histochemische Untersuchungen über das Phenomen der Pigmentdunkelung, Inaug. Dissert., Zurich, Gebr. Leemann & Co., 1939.

The former procedure, therefore, was the one that we adopted. It consisted of the following steps:

The sections of frozen skin were incubated in flasks containing air-tight rubber stoppers with perforations through which gases could be introduced and withdrawn from within the flasks. After the sections had been placed in the fluids within the flasks, the air was withdrawn from the flasks as completely as possible. Then nitrogen, after being filtered from a tank through a pyrogallol-alkali mixture to remove any trace of oxygen contamination, was driven through the flasks and their fluid contents for about half an hour. The outlets were then sealed and the flasks placed in an incubator for three hours. Dopa solution buffered to p_H 7.3, placed in a flask treated in the prescribed manner and kept at 37 C. for three hours regularly failed to show any change in color from its original water-clear appearance.

This procedure with dopa was carried out as a control in all experiments. This was the criterion that we had accepted as proof of the presence of an oxygen-free atmosphere.

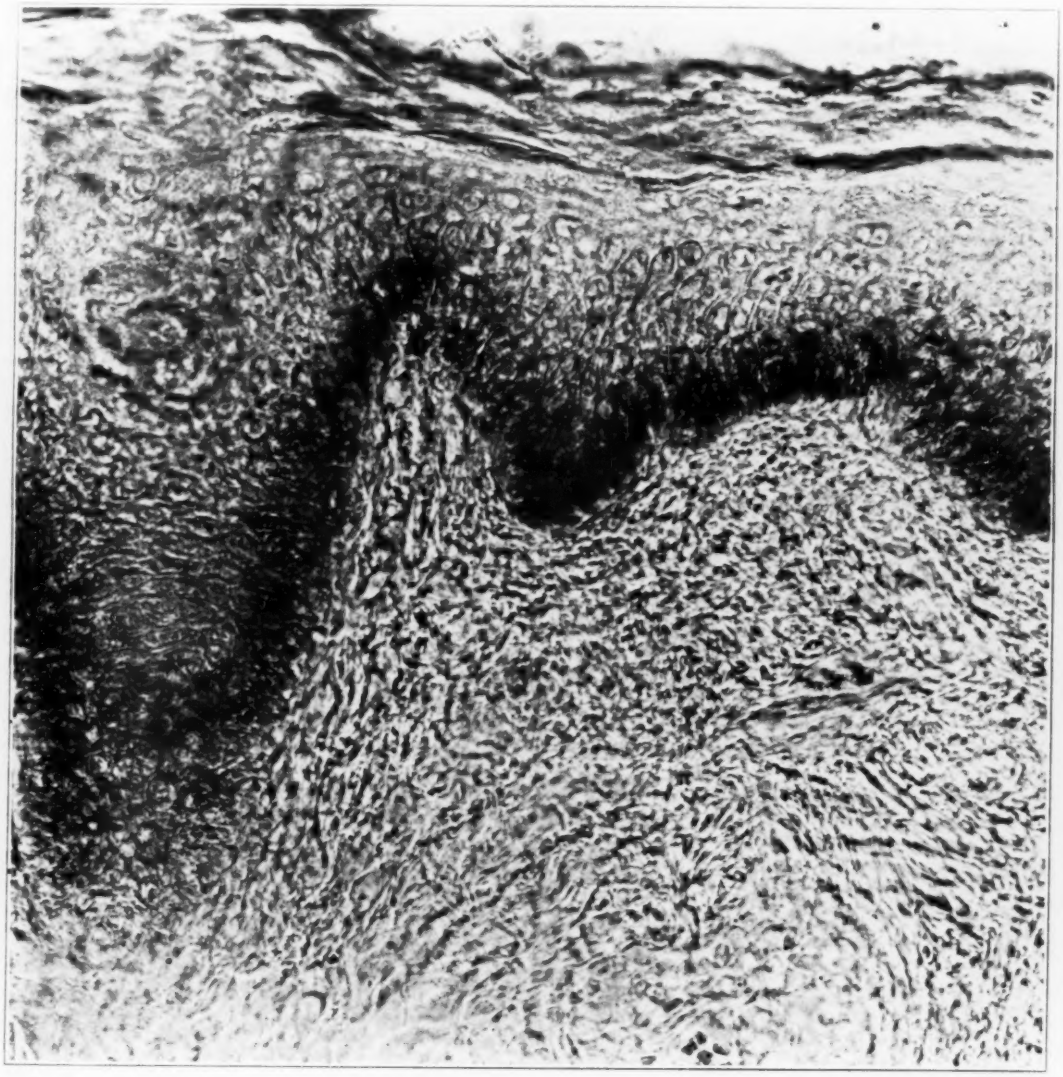

Fig. 2.—The pigment picture in the epidermis after incubation of the section in isotonic solution of sodium chloride in an atmosphere of air.

All the experiments included in our first report were repeated according to this procedure. The results, both quantitatively and qualitatively, paralleled those previously reported.

We offered no photomicrographs in our first report. Here we present seven, all of the same piece of tissue, in substantiation of the statement in the preceding paragraph.

The first three photomicrographs apply only to the types of experiment followed by all previous investigators of the Meirowsky phenomenon. The remaining four reveal the effects of protoplasmic poisons on pigmentation and constitute our original contribution to the experimental data dealing with the phenomenon.

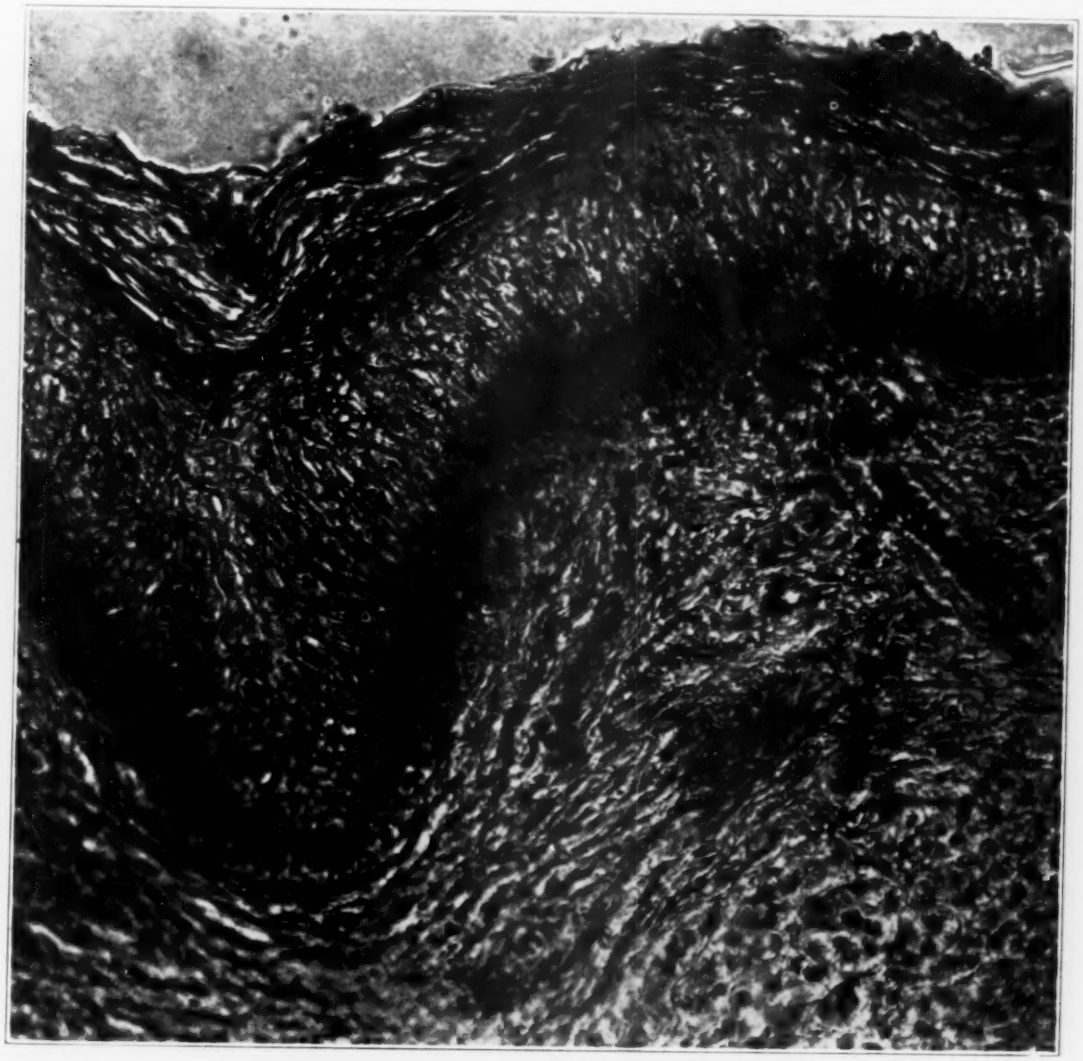

Fig. 3.—The pigment picture in the epidermis after incubation of the section in isotonic solution of sodium chloride in an atmosphere of nitrogen.

The conclusion is inescapable. The original Meirowsky phenomenon and our reported variations in eliciting it do not depend on an external source of oxygen for their production.

Miescher and Minder further reported [2] that sections of skin in which increased pigmentation has occurred as a result of incubation in air could be decolorized by reducing agents, and that the pigment could be reestablished by a repetition of the incubation in air. They could effect this cycle for a variable number of times, but finally the pigmented phase would become fixed and irreversible. From all these observations relative to the part atmospheric oxygen plays in the production of the Meirowsky phenomenon, they came to the following conclusions: (1) that this phenomenon of an increased number and size of melanin particles occurring in the epidermis from the basal layer up to the horny layer is simply a result of oxygenation of particles already formed; (2) that melanin exists with all degrees of oxygen saturation; (3) that the size and depth of the

color particle are determined by its state of oxygen saturation (the less the oxygenation, the smaller the particle and the paler the color); (4) that complete oxygen saturation is the stable form; (5) that the phenomenon therefore represents only the exerting of an influence on the size and color of the melanin particles already present, through further saturation of the particles with oxygen, and last (6) that since new

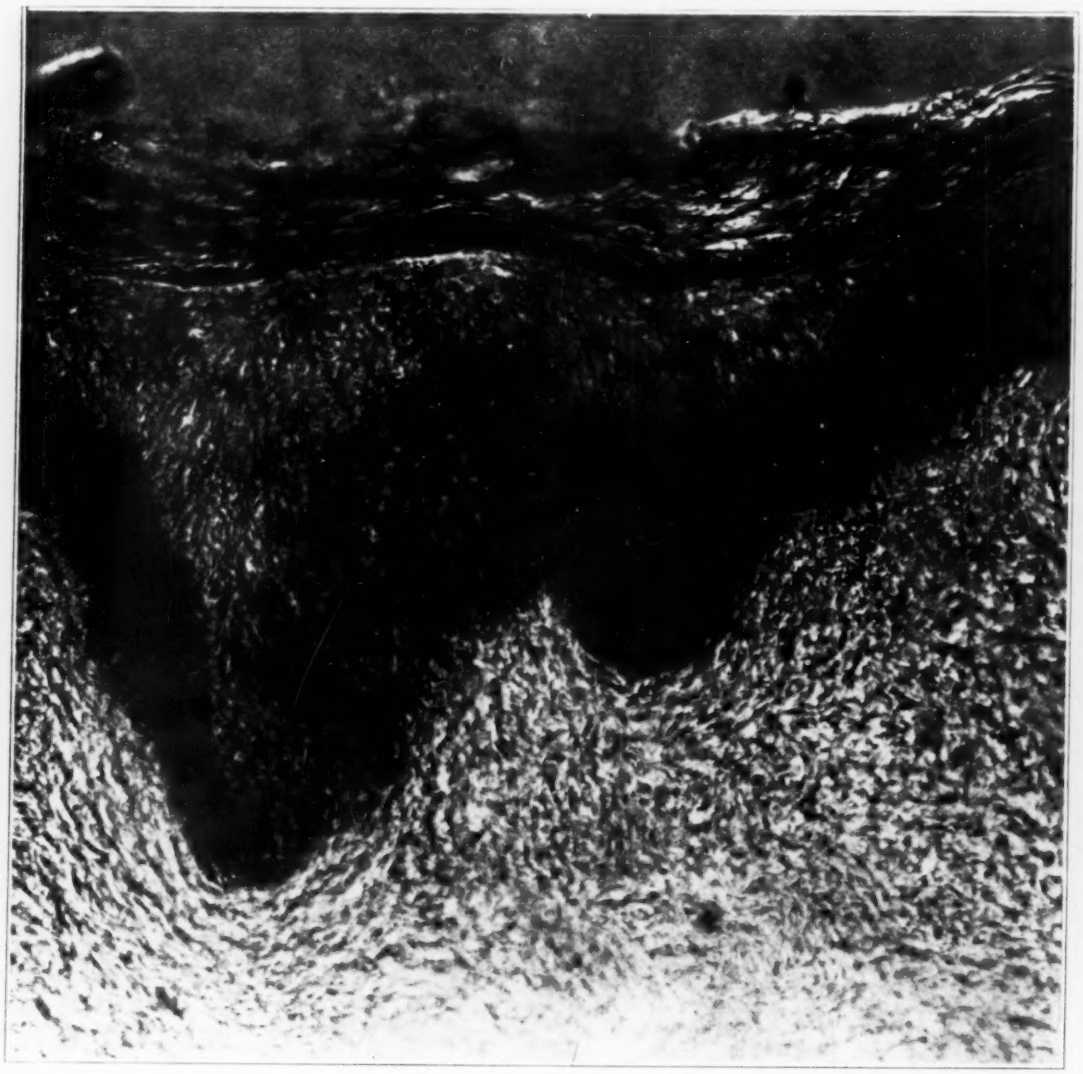

Fig. 4.—The pigment picture in the epidermis after incubation of the section in 0.1 per cent potassium cyanide solution buffered to p_H 9 in an atmosphere of air.

pigment production is not involved, the phenomenon has no bearing on the dopa theory of Bloch, and they therefore preferred to call it a darkening phenomenon.

What becomes of these conclusions if one rejects their experimental basis, namely, the presence of atmospheric oxygen in order to produce the phenomenon? I pose this question in this form because in all likelihood oxidation by means of acceptance of oxygen underlies the production of the phenomenon of Meirowsky. The conclusions of Miescher and Minder must be modified, in my opinion, to satisfy and offer an explanation for my experimental results, to wit: (1) the production of the phenomenon without the aid of atmospheric oxygen and (2) the accentuation of the pigmentary increase by the use of protoplasmic poisons.

I offer an explanation in the following terms: The Meirowsky phenomenon results from (1) the production of more melanin *de novo* in cells

of the epidermis having the capacity normally to form melanin (basal layer) and (2) the enlargement of all the existing particles by means of oxygen saturation through the making available within the cells of ionic oxygen throughout the epidermis. The availability of this oxygen is experimentally achieved by reducing or destroying the functional capacity of the cytochrome system of the cell (primary respiratory chain), which system has a primary call on all available oxygen. The damage to the cytochrome system is produced naturally (1) through death (cadaver skin), (2) by moribundity (excised skin) and experimentally (3) through other denaturing processes (skin submitted to freezing, hardening with solution of formaldehyde, poisoning with cyanides or fluorides). One may conceive of the pigment-forming system as functioning in the integrated skin under the handicap of a deficient supply of oxygen, which supply may be augmented by any local or systemic change calculated to upset the oxygen balance in the pigment-forming cell. Apparently, ultraviolet rays as a local factor and the aging process as a systemic one can be relied on to produce conditions conducive to increased pigment production.

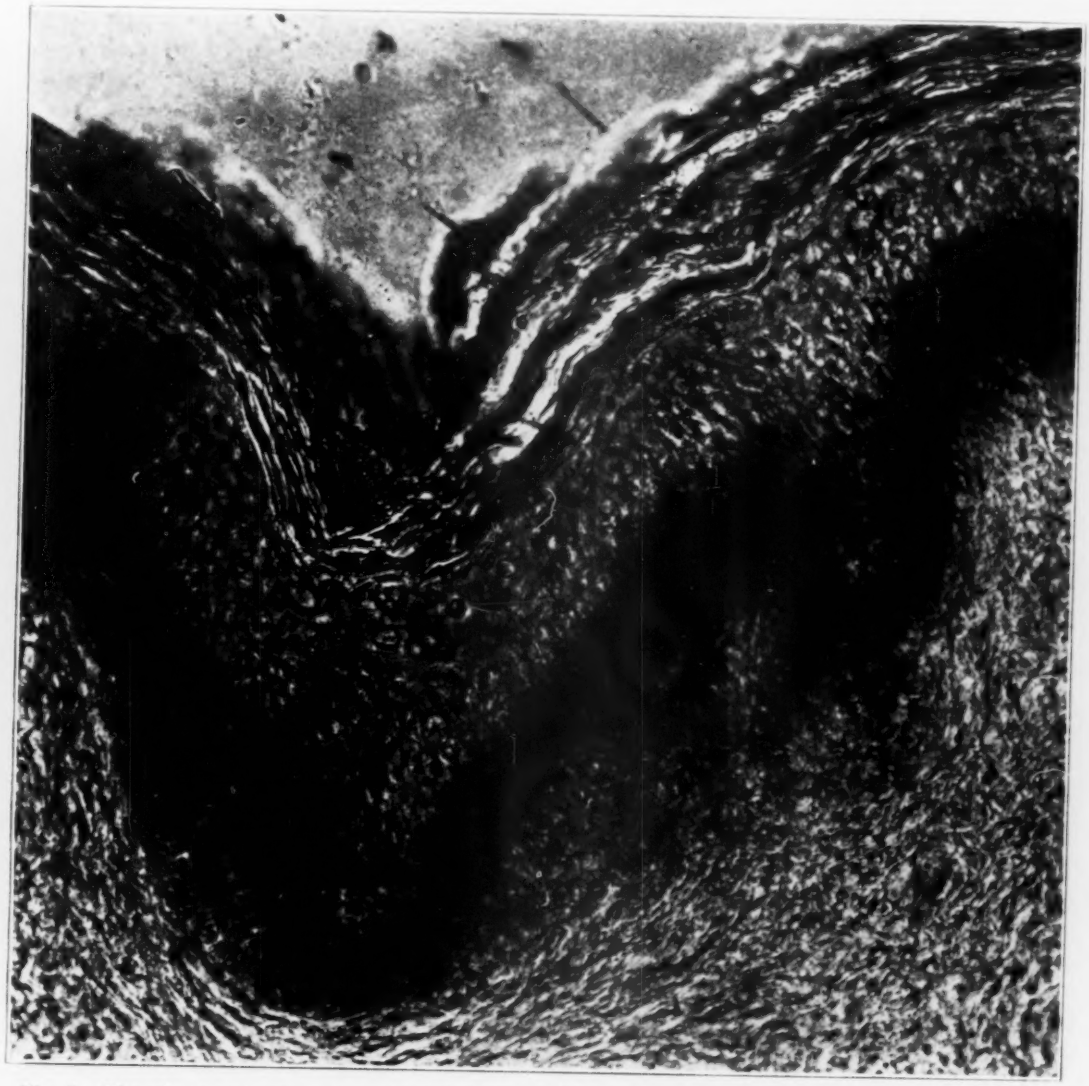

Fig. 5.—The pigment picture in the epidermis after incubation of the section in 0.1 per cent potassium cyanide solution buffered to p_H 9 in an atmosphere of nitrogen.

The adherents to the Bloch theory of melanin production have given the Meirowsky phenome-

non its most extensive probing. This suggests that an acceptance of the phenomenon apparently offers a forceful challenge to the dopa theory. Essentially, this theory has two indispensable features: an enzymatic process of converting premelanin into melanin and a specific character of the enzyme. Miescher and Minder, in asserting that the Meirowsky phenomenon is simply an effect of oxygen saturation of melanin particles, tried to sever the phenomenon from the experimental data relevant to the Bloch theory, on the assumed ground that the nonenzymatic process involved could not account for the production of new pigment. They were probably strengthened in this belief by the presentation of melanin particles in great number in layers of the epidermis above the basal layer, areas in which new-formed melanin does not physiologically occur. They refused to consider that the many new particles appearing in the basal layer, as revealed by the Meirowsky phenomenon, might be melanin *de novo* in cells capable of forming new pigment and that there was no inherent contradiction between the acceptance of their explanation for the presence of melanin in the upper layers of the epidermis and the belief in a new production of melanin in the basal layer under the same experimental conditions.

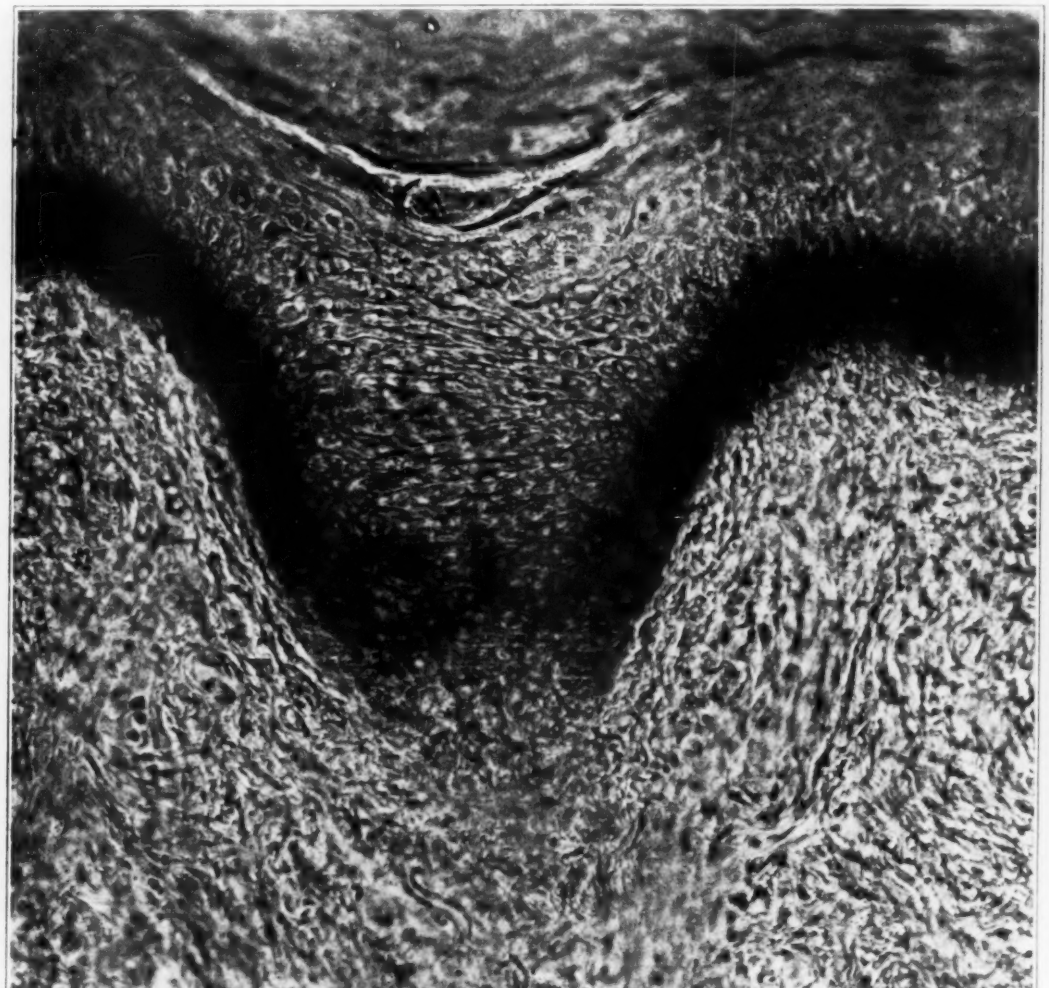

Fig. 6.—The pigment picture in the epidermis after incubation of the section in 0.1 per cent sodium fluoride buffered to p_H 9 in an atmosphere of air.

The explanation of the Meirowsky phenomenon by a completely nonenzymatic process becomes less tenable, to state it conservatively, if one accepts my reports of the elicitation of the phenomenon in an oxygen-free atmosphere. For under these conditions the necessity for an enzymatic process in its production becomes highly probable. Instead, then, of rejecting these data as pertinent to the Bloch theory, I must conclude that they are relevant. In this regard the phenomenon of Meirowsky becomes another step in solving the problem of the formation of pigment generally and the dopa theory particularly. The Bloch theory has yet to be disproved. Indeed, from many sources confirmation of several of its features is established. Further investigation of the Meirowsky phenomenon may serve to strengthen the Bloch theory rather than to refute it. But first the experimental facts must be amplified.

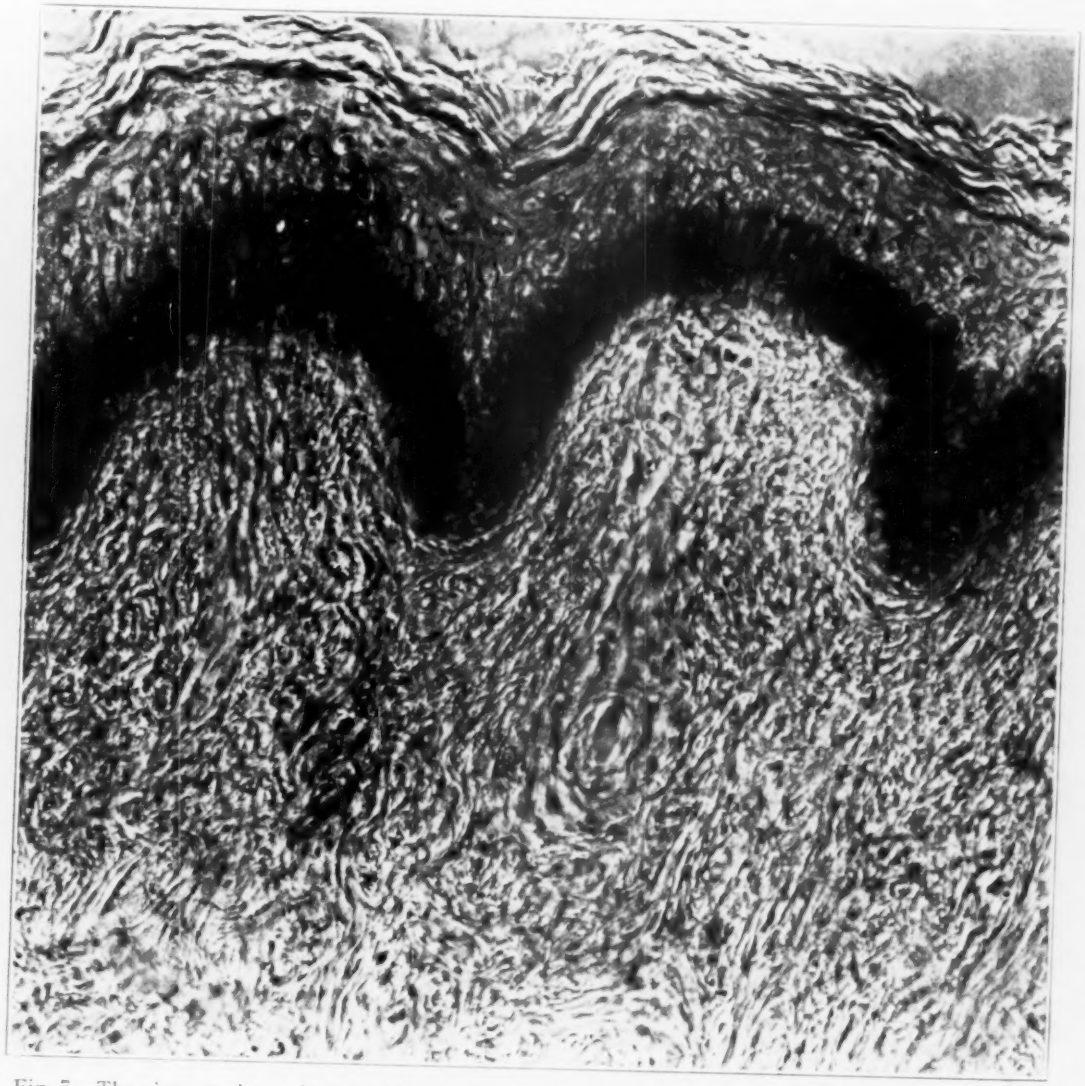

Fig. 7.—The pigment picture in the epidermis after incubation of the section in 0.1 per cent sodium fluoride buffered to p_H 9 in an atmosphere of nitrogen.

CUTANEOUS TUMORS OF VON RECKLINGHAUSEN'S DISEASE (NEUROFIBROMATOSIS)

REPORT OF A HISTOLOGIC STUDY, WITH SPECIAL REFERENCE TO NERVE FIBERS AND THE BODIAN STAIN *

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We wish to report a histologic study of the cutaneous tumors in 15 typical cases of von Recklinghausen's disease (neurofibromatosis) seen in the section on dermatology and syphilology of the Mayo Clinic. The purpose of the study is an attempt to assist in clarifying present conflicting opinions regarding the fibromatous, or neural, origin of these tumors.

This study involves chiefly the demonstration of individual nerve fibers in the tumors. For this purpose, an activated strong protein silver (protargol) stain was utilized. This method, first described by Bodian,[1] is a simple procedure for staining mounted paraffin sections of the central and peripheral nervous systems with reduced silver. It produces uniform, sharp, specific staining of peripheral nerve fibers in contrast to the fainter, grayish purple reticulum and coarser, lighter-staining collagen fibers, which are more readily distinguishable than they are when the usual argentophilic technic is employed. The procedure is as follows:

1. Impregnation with silver. A 1 per cent solution of strong protein silver (protargol) containing 4 to 6 Gm. of copper per 100 cc. is prepared, and the sections are immersed in this solution for twelve to forty-eight hours at 37 C. The sections then are removed and washed with distilled water.

2. Reduction with hydroquinone. The sections are permitted to stand for ten minutes in a solution of 1 Gm. of hydroquinone and 5 Gm. of sodium sulfite in 100 cc. of distilled water. They then are washed with distilled water.

3. Toning with gold. A 1 per cent solution of gold chloride in distilled water containing 3 drops of glacial acetic acid per 100 cc. is prepared. Sections are immersed for five to ten minutes in this solution. They then are washed with distilled water.

4. If sections do not have a light purple color, they are placed in a 2 per cent solution of oxalic acid until a purplish tinge appears (usually five to ten minutes). They then are washed with distilled water.

5. Removal of residual silver salts. The sections are immersed in a 5 per cent solution of sodium thiosulfate for five to ten minutes. They then are washed thoroughly in distilled water, dehydrated and mounted in balsam.

Von Recklinghausen's disease is a disease of varied clinical manifestations. The most frequent and almost constant feature observed is the presence of multiple cutaneous tumors, the so-called mollusca fibrosa, which vary exceedingly in extent and size, are irregularly distributed over the skin and show no evidence of localization corresponding to that of the peripheral nerves. In addition, pigmentary disturbances of the skin are numerous and frequently precede the appearance of the tumors in the form of various-sized macules and plaques. There may be tumors of the central and peripheral nervous systems and of the viscera, associated anomalies of the bones and possibly endocrine disturbances. Low standards of physical and mental development in patients with this disease have been reported by Charpentier [2] and other authors.[3]

The gross anatomic characteristics of this condition were first described by R. W. Smith [4] in

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This paper is an abridgment, with additions, of thesis submitted by Dr. McNairy to the Faculty of the Graduate School of the University of Minnesota in partial fulfilment of the requirements for the degree of M.S. in Dermatology and Syphilology.

1. Bodian, D.: A New Method for Staining Nerve Fibers and Nerve Endings in Mounted Paraffin Sections, Anat. Rec. **65**:89-97 (April) 1936.

2. Charpentier, J.: Maladie de Recklinghausen et psychose périodique, Encéphale **2**:460-465 (Oct. 20) 1910.

3. Ormsby, O. S., and Montgomery, H.: Diseases of the Skin, ed. 6, Philadelphia, Lea & Febiger, 1943. Levin, O. L., and Behrman, H. T.: Recklinghausen's Disease: Its Elusive Manifestations and Internal Relations, Arch. Dermat. & Syph. **41**:480-502 (March) 1940.

4. Smith, R. W.: A Treatise on the Pathology, Diagnosis, and Treatment of Neuroma, Dublin, Hodges & Smith, 1849.

1849. Virchow,[5] in 1863, was the first to study the tumors in detail. He classified them as true or false neuromas. He said that the true neuromas contained nerve cells and nerve fibers while the false neuromas arose from the connective tissue of the sheaths of the nerves and did not contain nerve cells or nerve fibers. In 1882, von Recklinghausen[6] called attention to the widespread nature of the disease. He contended that the tumors were chiefly fibrous and arose from mature connective tissue of the nerve sheaths. He said that some contained small to large numbers of nerve fibers. In 1910, Verocay,[7] who studied tumors of neurofibromatosis with the van Gieson-Weigert and the Heidenhain hematoxylin and eosin stains, reported the presence of long wavy fibers, allegedly nerves, which, he contended, proved that the tumors have a peculiar nerve tissue structure. The cellular elements were assumed to be immature cells of the sheath of Schwann, although he did not report the presence of mature nerve cells in the tumors. He therefore used the name "neurinoma" in an attempt to indicate that the tumors had the structure of nerve tissue rather than of connective tissue. From that time to the present, two schools of thought have developed regarding the origin and nature of these tumors. The theory proposed by Mallory,[8] Penfield[9] and others,[10] namely that the tumors arise from connective tissue, designates the fibroblast as the type cell of these tumors and is based largely on the identification of collagen, fibroglial and elastic tissues, which these investigators considered specific products of fibroblasts. The Schwannian theory, proposed by Masson[11] and others,[12] which eventuated from the morphologic and cultural studies of experimentally produced "schwannomas" in the cut sciatic nerve of the rabbit, implies that the type cell is of ectodermal origin.

These schools of thought have been formulated largely on the basis of histologic studies of tumors of the peripheral nerves, including those of von Recklinghausen's disease. Microscopically these tumors are similar to other nerve tumors, with an irregularly interwoven reticulated appearance, palisading of the nuclei and whorl arrangement superimposed. Neuropathologists generally describe two types of neurofibromas: one the solitary and the other the multiple; the latter type is associated with the other stigmas of von Recklinghausen's disease. Multiple neurofibromas are believed by some authors to differ from solitary neurofibromas histologically and to be a separate entity clinically. Penfield[9] pointed out that axis-cylinders coursed through multiple neurofibromas and that these tumors, therefore, were true neurofibromas. In the solitary tumor, however, the axis-cylinders of the nerve bundle from which the tumor arose were in the capsule and not within the tumor substance; as a result, the tumor was a perineural fibroblastoma. Kernohan[13] said that he had had much difficulty in distinguishing these types of tumors but that as a rule axis-cylinders are encountered more commonly within the tumors of von Recklinghausen's disease than they are within solitary neurofibromas.

The histologic picture of the cutaneous tumors, however, is less definite. They are sharply circumscribed and have a structure distinct from that of the adjacent cutis. They are described as being composed of peculiar nucleated bands and pale, fine, spindle-shaped fibrillae resembling those seen in nerve tissue. Absence of elastic fibers has been noted. The presence of nerve fibers has been reported by some authors but has been denied by others. Gans[14] demonstrated minute nerve fibers in the tumors by using Van

5. Virchow, R.: Die krankhaften Geschwülste, Berlin, A. Hirschwald, 1863-1867.

6. von Recklinghausen, F. D.: Ueber die multiplen Fibrome der Haut und ihre Beziehung zu den multiplen Neuromen. Festschrift zur Feier des fünfundzwanzigjährigen Bestehens des pathologischen Instituts zu Berlin, Berlin, A. Hirschwald, 1882.

7. Verocay, J.: Zur Kenntnis der Neurofibrome, Beitr. z. path. Anat. u. z. allg. Path. **48**:1-69, 1910.

8. Mallory, F. B.: The Type Cell of the So-Called Dural Endothelioma, J. M. Research **41**:349-364 (March) 1920.

9. Penfield, W.: Tumors of the Sheaths of the Nervous System, Arch. Neurol. & Psychiat. **27**:1298-1309 (June) 1932.

10. Rhoads, C. P., and Van Wagenen, W. P.: Observations on the Histology of the Tumors of the Nervus Acusticus, Am. J. Path. **4**:145-151 (March) 1928. Bailey, P., and Herrmann, J. D.: The Rôle of the Cells of Schwann in the Formation of Tumors of the Peripheral Nerves, ibid. **14**:1-37 (Jan.) 1938.

11. Masson, P.: Experimental and Spontaneous Schwannomas (Peripheral Gliomas), Am. J. Path. **8**:367-415 (July) 1932.

12. Murray, M. R.; Stout, A. P., and Bradley, C. F.: Schwann Cell Versus Fibroblast as the Origin of the Specific Nerve Sheath Tumor: Observations upon Normal Nerve Sheaths and Neurilemomas in Vitro, Am. J. Path. **16**:41-60 (Jan.) 1940. Tarlov, I. M.: The Origin of Perineural Fibroblastoma, ibid. **16**:33-40 (Jan.) 1940.

13. Kernohan, J. W.: Tumors of the Spinal Cord, Arch. Path. **32**:843-883 (Nov.) 1941.

14. Gans, O.: Histologie der Hautkrankheiten; die Gewebsveränderungen in der kranken Haut unter Berücksichtigung ihrer Entstehung und ihres Ablaufs, Berlin, Julius Springer, 1925, vol. 2.

Gieson's connective tissue stain. Friedlander [15] noted, in a single case, nerve fibers running parallel to the blood vessels and crossing and recrossing each other. Kriege, [16] Ehrmann [17] and other authors [18] also mentioned the presence of such fibers. On the other hand, Marie [19] could find no evidence of nerve fibers in the cutaneous tumors removed from a cadaver, although a few were thought to be seen in tumors removed from the patient during life. Gottheil [20] was unable to demonstrate nerve fibers in 3 cases studied, and Anderson,[21] examining a number of histologically representative tumors in a typical case of neurofibromatosis, failed to demonstrate nerve tissue of any kind in the cutaneous lesions. Stout and his associates [22] said, "Many of the fibromata mollusca, which are such an impressive feature of von Recklinghausen's disease, prove on histological examination to have the morphology of simple fibromata, and a connection with nerves cannot be demonstrated."

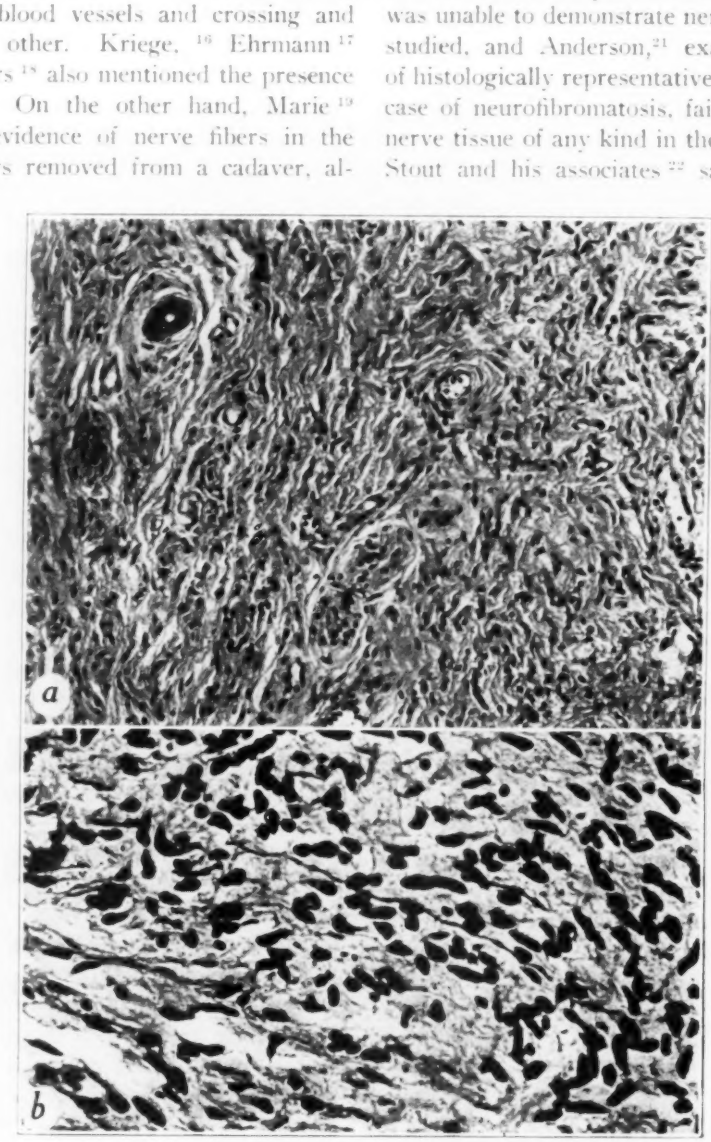

Fig. 1.—(*a*) Typical cutaneous tumor of von Recklinghausen's disease in case 1; section stained with hematoxylin and eosin (× 160). (*b*) Nerve fibers in tumor in case 1; section stained with Bodian's stain (× 420).

15. Friedlander, D.: Report of a Case of Multiple Neurofibromata, with a Review of the Subject, Based on Two Hundred and Sixty-Two Cases Reported in the Literature, J. Cutan. Dis. **28**:497-505 (Oct.) 1910.

16. Kriege, H.: Ueber das Verhalten der Nervenfasern in den multiplen Fibromen der Haut und in den Neuromen, Virchows Arch. f. path. Anat. **108**:466-493 (June) 1887.

17. Ehrmann, S.: Anatomischer und klinischer Beitrag zur Kenntnis der Recklinghausenschen Krankheit, Arch. f. Dermat. u. Syph. **129**:498-515, 1921.

18. Ebert, M. H.: Recklinghausen's Disease with Demonstration of Nerve Fibers in a Tumor, Arch. Dermat. & Syph. **42**:190 (July) 1940.

19. Marie, cited by Keen and Spiller.[23]

20. Gottheil, in discussion on Ravogli, A.: Fibroma Molluscum, or Universal Neurofibromatosis, J. Cutan. Dis. **29**:79 (Feb.) 1911.

21. Anderson, L. H.: Generalized Neurofibromatosis, with Report of a Case, J. A. M. A. **74**:1018-1021 (April 10) 1920.

22. Stout, A. P.; Laidlaw, G. F., and Haagensen, C. D.: Tumors of Peripheral Nerves, A. Research Nerv. & Ment. Dis., Proc. **16**:417-439, 1937.

The cutaneous lesions selected for study included soft, pedunculated papillomatous tumors as well as bluish, domelike ones and, in several instances, pigmented café-au-lait spots. Because of the frequent suggestion by other authors[23] that large tumors might show no evidence of nerve fibers as a result of pressure atrophy or degenerative changes, small ones were selected wherever a choice was possible. Since in several instances the material for study was obtained as the result of excision for cosmetic or therapeutic reasons, tumors of various sizes and ages were included; hence the most desirable specimens were not obtained in all cases. Numerous sections were made of the tumors. In some instances serial sections were studied in an attempt to prove or disprove the presence of nerve fibers. In addition to the Bodian stain, comparative studies were made with hematoxylin and eosin, Maresch-Bielschowsky stain, Gros-Bielschowsky stain and a modification of Masson's trichrome stain (figs. 1, 2 and 3). In several cases, Orlandi's stain and cresyl violet also were used.

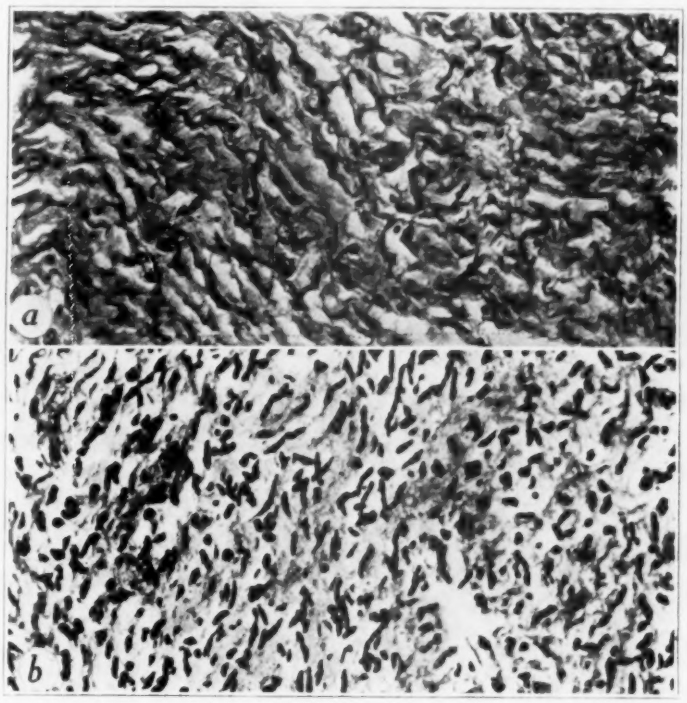

Fig. 2.—(*a*) Coarse wavy reticulum fibers (*Gitterfasern*) demonstrated with Maresch-Bielschowsky technic (× 260). (*b*) Section showing irregular, lozenge-shaped, densely staining nuclei; section stained with the Gros-Bielschowsky method (× 260).

REPORT OF CASES

We shall report 2 of the 15 cases that form the basis of this report.

CASE 1.—A white youth aged 18 years had noted the gradual appearance of pigmented macular areas and multiple soft bluish tumors, scattered principally over the trunk, since the age of 9 years. Histologic study of a lesion excised from the right flank revealed a circumscribed oval tumor lying in the midcutis and composed of numerous cells with irregular, elongated, densely staining nuclei and lighter, somewhat granular, ill-defined cytoplasm (fig. 1*a*). The cells terminated in long, tapering, unipolar and bipolar processes. The arrangement of the cells was irregular, and there was no evidence of the formation of whorls. Between the cells was a spongy, ill defined reticular substance. Throughout the tumor, especially in the outer third, were numerous dilated vessels and lymph spaces, nerve bundles and in one area sebaceous glands and sweat glands. The sweat glands were flattened, were separated by the tumor substance and showed evidence of degenerative changes. Individual nerve fibers, densely stained, sharply demarcated, somewhat larger than the cell processes and independent of cell bodies, were readily identified with the Bodian stain in several areas in the tumor substance (fig. 1*b*). None were identified in association with nerve bundles or dermal appendages.

CASE 2.—A married woman aged 42 years presented a typical picture of von Recklinghausen's disease with multiple pigmented macules, "café au lait" spots and cutaneous tumors. A specimen for biopsy was obtained on two occasions. One specimen was obtained from a pigmented plaque in the midscapular region; the other was obtained from a soft bluish tumor which was

23. Keen, W. W., and Spiller, W. G.: A Case of Multiple Neuro-Fibromata of the Ulnar Nerve, Am. J. M. Sc. **119**:526-539 (May) 1900. Ravogli, A.: Fibroma Molluscum, or Universal Neurofibromatosis, J. Cutan. Dis. **29**:71-78 (Feb.) 1911.

bladder-like and readily compressible into a hollow depression in the subcutaneous tissue. Histologically the pigmented plaque revealed little variation from the normal epidermis and cutis other than increased deposits of melanin in the basal layer and in a few dendritic cells in the epidermis. No evidence of nerve fibers or tumor cells could be demonstrated. The other specimen disclosed a typical histologic structure with cellular and reticular components similar to those found in case 1 and a retiform pattern penetrated by numerous dilated vessels and lymph spaces. Short, coarse, deeply stained, wavy nerve fibers were readily discernible in several sites, one near the center of the tumor, another laterally and adjacent to a small vessel (fig. 3*a*).

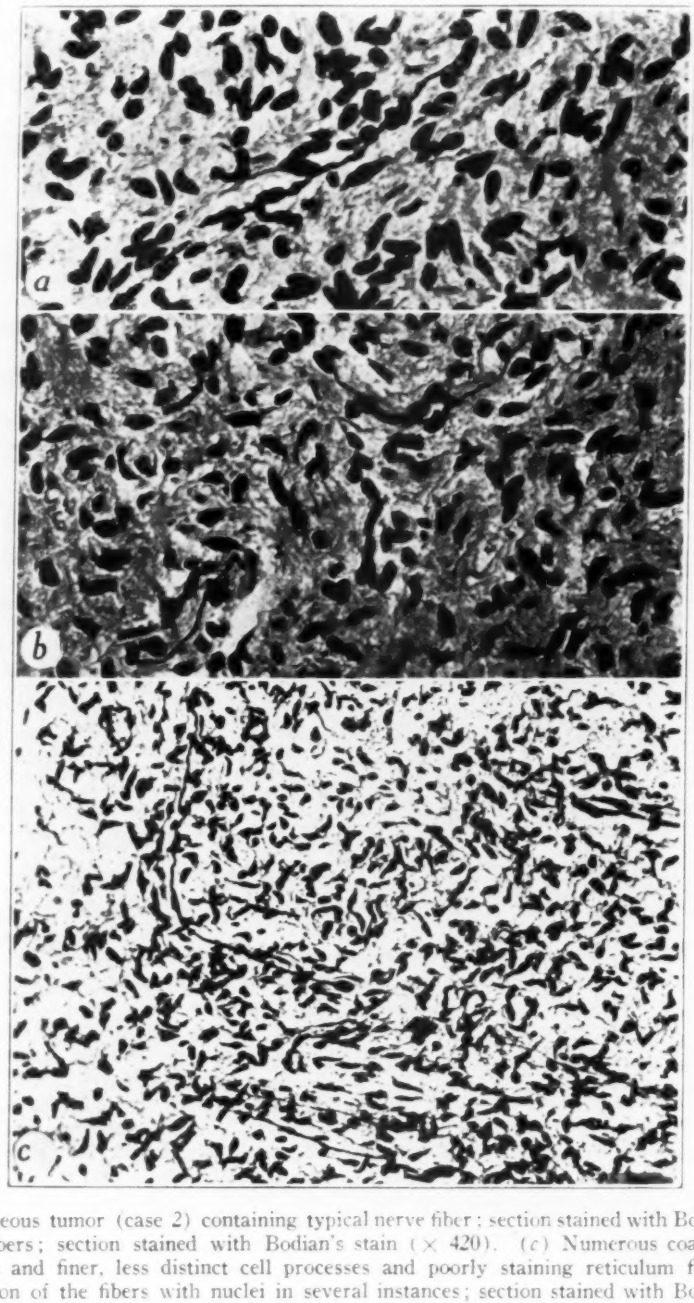

Fig. 3.—(*a*) Cutaneous tumor (case 2) containing typical nerve fiber; section stained with Bodian's stain (× 420). (*b*) Typical nerve fibers; section stained with Bodian's stain (× 420). (*c*) Numerous coarse, densely staining, irregular nerve fibers and finer, less distinct cell processes and poorly staining reticulum fibers; one may note the apparent association of the fibers with nuclei in several instances; section stained with Bodian's stain (× 260).

COMMENT ON HISTOLOGIC FINDINGS

The present study was prompted by the conflicting reports in the literature regarding the presence or absence of nerve fibers in the cutaneous tumors. Verocay's[7] belief that the long fibers he demonstrated in the cutaneous tumors were of nerve origin, for example, was contested by Penfield,[9] who by selective staining with silver carbonate demonstrated that they were connective tissue fibers. Most of the studies of such tumors have included only 1 case or, at most,

several cases. Various silver impregnation methods have been preferred by most investigators. Since reticulum fibers stain readily with the usual argentophilic methods, it is conceivable that this might be one source of confusion in the attempts to demonstrate nonmedullated nerve fibers. That the structures are readily distinguished by the Bodian stain is demonstrated by the accompanying photomicrographs (figs. 1 to 3).

In each of the 15 cases, nerve bundles were present, usually in the peripheral portions of the tumor. These bundles varied considerably in size and number. Their presence is not adjudged particularly significant from the standpoint of origin of the tumor, since normal dermis and corium are abundantly supplied with nerve structures. In 1 instance, individual nerve fibers were identified in the vicinity of a nerve bundle and extended toward the center of the tumor, thus suggesting their position as finer ramifications of a peripheral nerve. In several cases there were degenerative changes in the nerve bundles, with separation of the individual fibers and partial disappearance of myelin sheaths. These are degenerative changes associated with pressure atrophy, as described by Kurz [24] and other authors.[25]

In 12 cases we were able to demonstrate individual nonmedullated nerve fibers in the cutaneous tumors, generally toward the center of the tumor. They ranged from few to numerous and from short to long and were in all cases characteristic, densely stained and distinct, wavy or irregular and did not run parallel to the tumor cells or their processes. In most instances these fibers were entirely independent of other structures, but in several sections they were contiguous with tumor cells. It was impossible to demonstrate whether this was a definite protoplasmic relationship or whether the structures were merely superimposed.

In 3 cases, many of the tumor cells with large, lighter staining nuclei and exceedingly long, wavy, bipolar and monopolar processes bore a striking resemblance to nerve cells. Cresyl violet was utilized to demonstrate the absence of neurofibrils and Nissl bodies. The processes, which stimulated nerve fibers, were finer, were less distinct and stained unevenly, which gave them a granular appearance in contrast to the even, jet-black appearance of true nerve fibers. Kernohan [26] and other authors [27] have pointed out that degenerating tumor cells can simulate, and sometimes are difficult to distinguish from, nerve tissue. Since specimens in these cases were large, and hence old, and as degenerative changes were noted in some of the dermal appendages and other structures, it can be assumed that the cell processes in question were most likely a part of degenerating fibroblastic tumor cells.

In 3 cases, histologic examination revealed the typical architecture of cutaneous tumors of von Recklinghausen's disease but failed to reveal any structures resembling nerve fibers or cells.

Two cases are of particular interest in that the specimens purposely were obtained from soft, bluish, deep-seated cutaneous tumors. The palpating end of the finger was able to press these balloon-like lesions into and below the level of the skin. This produced the feeling of forcing the soft tumor through a sharply marginated ring, as in a ventral hernia. On withdrawal of the finger, the tumors assumed their original size and shape. Clinically, these lesions simulated a condition first reported by Schweninger and Buzzi [28] under the title of "Multiple Tumor-Like New Growths of the Skin." These authors described the insidious onset of balloon-like lesions. Some of the lesions were the color of natural skin; others were bluish or of a slate-tinted hue. In cases of this condition reported by other authors, the lesions have occurred both independently and in association with syphilis. Balloon-like atrophy may appear after the inflammatory lesions of dermatoses such as secondary syphilis, tuberculosis and leprosy. Anetodermia of Jadassohn,[29] while originating as a series of erythematous papules, results in similar atrophic, herniated tumor-like lesions. Clinically, these conditions might conceivably be confused with neurofibromatosis. Chargin and Silver,[30] in a review of the literature on macular atrophy of the skin, said that some of the cases reported by various authors were actually cases of von Recklinghausen's disease. Histologically

24. Kurz, cited by Takino.[25a]

25. (*a*) Takino, M.: Ueber die Recklinghausensche Krankheit, besonders ihre Beziehung zu den Hautnerven, Acta scholae med. univ. imp. in Kioto **14**:1-15, 1931. (*b*) Penfield, W.: Cytology and Cellular Pathology of the Nervous System, New York, Paul B. Hoeber, Inc., 1932, vol. 3.

26. Kernohan, J. W.: Personal communication to the authors.

27. Penfield, W., and Young, A. W.: The Nature of von Recklinghausen's Disease and the Tumors Associated with It, Arch. Neurol. & Psychiat. **23**:320-344 (Feb.) 1930.

28. Schweninger and Buzzi, cited by Chargin and Silver.[30]

29. Jadassohn: Ueber eine eigenartige Form von "Atrophia maculosa cutis," Arch. f. Dermat. u. Syph. **24**:342-358, 1892.

30. Chargin, L., and Silver, H.: Macular Atrophy of the Skin, Arch. Dermat. & Syph. **24**:614-643 (Oct.) 1931.

there should be no confusion. The balloon-like tumors of neurofibromatosis are new growths which definitely are circumscribed and have a characteristic architecture. The other diseases have no tumor structure, since they consist essentially of macular atrophy. The Schweninger and Buzzi type of lesions exhibits absence of elastic fibers in the affected regions, a slight increase of these fibers at the margins, round cell infiltration about the superficial blood vessels and dermal appendages and a general picture of cutaneous atrophy. These lesions are distinct from the balloon-like lesions of von Recklinghausen's disease. Anetodermia of Jadassohn shows histologic changes essentially the same as those of acrodermatitis chronica atrophicans, according to the original description, although the infiltrate in the former lies deeper and does not form a border zone between the epidermis and upper parts of the cutis and the rete ridges usually are preserved.

In 2 instances specimens for biopsy were obtained from café-au-lait spots, since some authors [31] have suggested that these pigmented spots may be the forerunners of subsequent tumor development in abortive types. Histologic examination failed to disclose any evidence of tumor cells or nerve structures in the underlying cutis. The sole abnormality consisted of increased deposits of melanin in the basal and dendritic cells of the epidermis.

DIFFERENTIAL DIAGNOSIS

Clinical features are usually sufficiently distinctive to enable one to distinguish von Recklinghausen's disease from other types of dermatosis. The histologic picture is characteristic in cases in which the clinical picture might be in doubt. The group of macular atrophies, including tumor-like new growths of Schweninger and Buzzi and anetodermia of Jadassohn, already has been distinguished from von Recklinghausen's disease. Fibroma molle, or cutaneous tags, single or multiple, as well as cutaneous tags of the neck (Templeton [32]) and fibroma molluscum gravidarum (Brickner [33]), all have a similar histologic appearance and are readily distinguished, since the involved portion of the cutis has an indefinite, loose, spongy areolar arrangement with a slight decrease in elastic tissue fibers. There is no evidence of neurofibroma. Keloids are usually readily designated grossly. The microscopic picture might be confused with that of a neurofibroma, especially in the earlier lesions, in which cellular elements are more prominent. There are, however, coarse collagenous fibers, arranged in bundles which cross and interweave at various planes, and oval cells with large vesicular nuclei. These cells are not so elongated as the cells of a neurofibroma and they do not stain so deeply with special silver stains. There also is an absence of cell processes and nerve fibers. Neuromas are usually solitary lesions or are few in number. They are tender and firm, have a purplish to pink hue, are fixed in the deeper layers of the cutis and are histologically distinctive, with a mixture of fine connective tissue and numerous medullated and nonmedullated nerve fibers. They do not contain neurofibromatous cells. Ganglioneuromas can be readily ruled out by the characteristic ganglion cells, which are not seen in cutaneous neurofibromas. Glomus tumor, in addition to being in most instances a solitary painful nodule usually situated on an extremity, has a characteristic histologic picture with central dilated arteriovenous anastomosis, hyperplasia of epithelioid-like cells and encapsulating nonmedullated nerve fibers in various proportions.

SUMMARY

This paper is based on a histologic study of 15 cases of von Recklinghausen's disease (neurofibromatosis). The study involved chiefly a search for nerve fibers with Bodian's method of staining. In 12 cases, nonmedullated nerve fibers were clearly demonstrated. In 3 cases, nerve fibers could not be identified. The presence of nerve fibers in the majority of cutaneous neurofibromas studied is supportive evidence that these lesions are true neurofibromatous structures which arise from the peripheral nerves and their supporting structures and that these nerve fibers are peripheral extensions of the nerve bundles which have been included in the expanding nerve sheath tumors. The cutaneous tumors of von Recklinghausen's disease have a definite histologic architecture and are thus readily distinguished from other cutaneous tumors or from the tumor-like macular atrophy of Schweninger and Buzzi. Pigmented macular lesions seen in neurofibromatosis were studied in 2 cases. No histologic evidence of an underlying neurofibromatous structure or of an increase in nerve fibers could be demonstrated.

31. Weber, F. P.: Cutaneous Pigmentation as an Incomplete Form of Recklinghausen's Disease, with Remarks on the Classification of Incomplete and Anomalous Forms of Recklinghausen's Disease, Brit. J. Dermat. **21**:49-53 (Feb.) 1909. Soldan: Ueber die Beziehungen der Pigmentmäler zur Neurofibromatose, Arch. f. klin. Chir. **59**:261-296, 1899.

32. Templeton, H. J.: Cutaneous Tags of the Neck, Arch. Dermat. & Syph. **33**:495-503 (March) 1936.

33. Brickner, S. M.: Fibroma Molluscum Gravidarum: A New Clinical Entity, Am. J. Obst. **53**:191-199 (Feb.) 1906.

CUTANEOUS DETERGENTS

EXPERIENCE WITH AN ETHER SULFONATE COMPOUND

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In 1939 a survey was made of 4,500 persons, including physicians, dentists, nurses, students, typists, mothers with young babies, childless housewives, mechanics and others, in order to determine the effect of soap as used in diverse occupations. Analysis of the replies to the varied but definite questions revealed that over 34 per cent of the group gave a history indicating that soap had caused dryness, scaling, itching or burning or some combination of these subjective and objective symptoms. Nurses, dental assistants, surgeons and housewives reported a higher percentage of ill effects from soap than did clerks or typists. About 13 per cent of the persons reporting disturbances were men. Dryness was the most common complaint, and over a third of those reporting dryness also reported associated itching and scaling. By far the most common substitute for soap was some kind of cosmetic cream. Ninety per cent of a group of several hundred persons who reported that they stopped using soap were relieved of symptoms. About 70 per cent of this group resumed its use, and approximately 40 per cent of these reported a recurrence of trouble within a few days or weeks.

Accurate statistical information is difficult to obtain from the subjective reports of patients untrained to evaluate etiologic factors, but it appears from a study of the results of this survey that a substantial percentage of persons have cutaneous irritation from the use of soap.

Many authorities [1] in the fields of dermatology and of industrial medicine state the belief that the use of soap may initiate, aggravate or prolong a dermatitis and may be a predisposing factor in the development of cutaneous irritation or sensitization from other causes. Some investigators [2] reported their belief that the alkali content is the cause, while others [3] expressed the opinion that the fatty acids commonly present in soap are responsible. A third explanation has been offered,[3a] which is that the irritating properties of the fatty acids are intensified in the presence of alkali and that this is the *modus operandi* in the development of the irritation. A comprehensive study was made by Blank [3a] to determine the effects of soap on the skin. His paper presents data on the action of fatty acids applied to the skin during experiments with controlled hydrogen ion concentrations, and he believed that this action helped to explain how irritation was produced. Although aqueous solutions of the commercial soaps tested showed a p_H value of between 10 and 11, he concluded that irritation resulted from neither the fatty acids alone nor the alkali of the soap but from a combination of the two. His studies supplied abundant evidence that the use of soap during treatment of contact dermatitis was inadvisable because, even if the soap was not the etiologic agent, it nevertheless prolonged the irritation after the direct cause was eliminated.

It was reported [4] that the deposition of insoluble soap on the skin also might be provocative of cutaneous disturbance. These deposits result from the combining of the fatty acids, freed by hydrolysis, with the lime salts present in water,

1. (*a*) Schwartz, L.: Protective Ointments and Industrial Cleansers, M. Clin. North America **26**:1195 (July) 1942. (*b*) Klauder, J. V.; Gross, E. R., and Brown, H.: Prevention of Industrial Dermatitis, with Reference to Protective Hand Creams, Soap and the Harmful Role of Some Cleansing Agents, Arch. Dermat. & Syph. **41**:331-356 (Feb.) 1940. (*c*) Sensitivity to Soap, Queries and Minor Notes, J. A. M. A. **117**: 1662 (Nov. 8) 1941. (*d*) Jordon, J. W.; Dolce, F. A., and Osborne, E. D.: Dermatitis of the Hands in Housewives, ibid. **115**:1001 (Sept. 21) 1940. (*e*) Downing, J. G.: Care of the Skin, New England J. Med. **225**:85 (July 10) 1941. (*f*) Goldman, L.: Patch Test with Soaps, M. Bull. Univ. Cincinnati **7**:90 (Nov.) 1935. (*g*) Goldman, L.: The Skin Reactions of Infants and Children to Soaps, J. A. M. A. **108**:1317 (April 17) 1937. (*h*) Hazen, H. H.: Allergic Dermatoses, Arch. Dermat. & Syph. **18**:121 (July) 1928. (*i*) Sulzberger, M. B., in discussion on Jordon, Dolce and Osborne.[1d]

2. (*a*) Mayer, R. L.: Toxidermien, in Jadassohn, J.: Handbuch der Haut- und Geschlechtskrankheiten, Berlin, Julius Springer, 1933, vol. 4, pt. 2. (*b*) Chiego, B., and Silver, H.: The Effect of Alkalis on the Stability of Keratins, J. Invest. Dermat. **5**:95 (April) 1942.

3. (*a*) Blank, I. H.: Action of Soap on Skin, Arch. Dermat. & Syph. **39**:811 (May) 1939. (*b*) Chiego and Silver.[2b]

4. Whitcomb, F. C., and Arnold, L.: The Relationship Between Acid-Base Equilibrium and Endogenous Bacterial Flora of the Skin, J. Invest. Dermat. **4**:317 (Aug.) 1941.

especially hard water. Such deposits are resistant to the action of detergents and antiseptics and therefore may harbor active bacteria. According to Jones and Lorenz,[5] "The prevention of calcium soap formation in wash water and its deposition on skin, hair or clothing is an active prophylactic measure against staphylococcal skin infection."

In some instances dryness and scaling may be due not to the composition or the *p*H value of a detergent but to its defatting action. Furthermore, certain persons have a low threshold of tolerance to detergents due to the inability of their skin to buffer alkali properly or to excrete normal amounts of sebum. The skin under such conditions may conceivably succumb more readily to the influence of industrial irritants or allergenic substances, such as cosmetics and plant oils.

Soap is defined as a cleansing agent made by the action of alkali on fat or fat acids. All soaps, even neutral soaps, hydrolyze during the process of washing to develop a *p*H value in the neighborhood of 10 or 11. However, the normal cutaneous surface has a *p*H averaging 5.5.[6] Blank[7] showed that most readings determined with the glass electrode of the Beckman *p*H meter were within the range of 4.2 to 5.6.

If the cutaneous surface is tested with the Beckman *p*H meter after the use of soap and after rinsing and drying, it will usually be found to have a *p*H of from 8 to 10; and, varying with the person, an interval from fifteen minutes to a few hours will elapse before a value within the normal range is established by the physiologic action of the skin.

DESIRABLE PROPERTIES OF A CUTANEOUS DETERGENT

Before a discussion of substitutes for soap it is advisable for one to consider the following specifications for a satisfactory detergent:

1. When worked with water it should emulsify and peptize the surface layer mixture of excreted and accumulated fats that have been adsorbed by particles of soil, so that it can be dispersed from the skin by rinsing.

2. It should not interfere with the normal functions of the skin or damage the protective layers of the epidermis.

3. The hydrolyzed product should have a *p*H value that corresponds to that of the average normal skin.

4. No undesirable residual layer should be deposited on the skin.

5. It should cause no discomfort and should be pleasurable and agreeable to use in order that physicians prescribing its use may obtain the cooperation of patients.

6. It should produce suds. For some three centuries suds have been a symbol of cleansing efficiency. Suds do assist mechanically in dispersing soil. The development of suds indicates when sufficient detergent material has been applied. Inability to produce further suds is a rough index of the exhaustion of the detergent property. The disappearance of suds after repeated rubbing and rinsing demonstrates the removal of the detergent material.

DETERGENTS USED AS SUBSTITUTES FOR SOAP

A variety of agents have been used as substitutes for soap in an attempt to remove soil without producing undesirable subjective or objective symptoms. They may be grouped as follows: (1) water, the greatest value of which is to aid the detergent action of other agents; (2) substances that have an adsorbent action, such as wet cornmeal and colloidal clays, or adsorbent abrasives, such as pumice and sand; (3) materials, such as liquid petrolatum, petrolatum and cosmetic creams, that partially emulsify and detach accumulated fat and soil; (4) so-called neutral soaps, that emulsify, saponify and disperse accumulated fat and soil; (5) superfatted soaps; (6) sulfated oils (commonly but improperly called sulfonated oils),[8] that emulsify fats and when used with water disperse soil; (7) sulfated alcohols; (8) sulfated esters; (9) sulfonated ethers.

5. Jones, K. K., and Lorenz, M.: The Relation of Calcium Soaps to Staphylococcal Infections of the Skin, J. Invest. Dermat. **4**:69 (Feb.) 1941.

6. Sharlit, H., and Scheer, M.: Hydrogen-Ion Concentration of the Surface of the Healthy Intact Skin, Arch. Dermat. & Syph. **7**:592 (May) 1923. Memmesheimer, A.: Die N-Ionen Konzentration der Hautoberfläche, Klin. Wchnschr. **3**:2102 (Nov. 11) 1924. Levin, O. L., and Silvers, S. H.: The Reaction of the Skin and Its Secretions in Eczema: I. The Hydrogen Ion Concentration of the Skin Surface in Eczema, Arch. Dermat. & Syph. **25**:825 (May) 1932. These articles are cited in an article by Draize, J. H.: The Determination of the *p*H of the Skin of Man and Common Laboratory Animals, J. Invest. Dermat. **5**:77 (April) 1942.

7. Blank, I. H.: Measurement of *p*H of the Skin Surface, J. Invest. Dermat. **2**:67 and 75 (April) 1939.

8. (*a*) Lane, C. G., and Blank, I. H.: Cutaneous Detergents, J. A. M. A. **118**:804 (March 7) 1942. (*b*) Snell, F. D.: Surface-Active Agents, Indust. & Engin. Chem. **36**:1 (Jan.) 1943. (*c*) Epstein, S.; Throndsen, A. H.; Dock, W., and Tainter, M. L.: Possible Deleterious Effects of Using Soap Substitutes in Dentifrices, J. Am. Dent. A. **26**:1461 (Sept.) 1939. (*d*) Gershenfield, L., and Witlin, B.: Surface Tension Reducents in Bactericidal Solutions: Their in Vitro and in Vivo Efficiencies, Am. J. Pharmacol. **113**:215 (June) 1941.

OBJECTIONS TO THESE SUBSTITUTES

Water only partially dislodges and disperses soil and does not remove soil combined with fats or oils. Materials such as those mentioned in group 2 are poor detergents, causing dryness and epidermal loss. Those in group 3 only partially emulsify the sebum and grease adsorbed by the soil, and the resulting mixture can only be partially wiped from the skin. This does not constitute complete or pleasing detergency. Neutral soaps contain fatty acids that may cause irritation and that when hydrolyzed develop a definite alkaline reaction. As with other soaps, when they are used with tap water (especially hard water) insoluble calcium and magnesium salts are formed and deposited on the skin, hair and bathing equipment ("bathtub ring"). Superfatted soaps are subject to the foregoing criticism. Sulfated oils tend to be drying and have been reported to be irritating in an appreciable percentage of cases.[9] Furthermore, they do not produce suds and otherwise are esthetically unsatisfactory.[10] Sulfated alcohols and sulfated esters are good detergents and do produce suds but are objectionable, as are the sulfated oils, because of their drying properties and the irritation that may be produced by the products of hydrolysis. Sulfonated ethers merit special consideration and will be discussed at length in the following section.

SULFONATES CONTRASTED WITH SULFATES

If a true soluble alkyl sulfate is mixed with water, it will produce sulfuric acid. All the commercial alkyl sulfates tested gave a *p*H value above 7, but when they were mixed with water the *p*H drifted to a decidedly acid level. This hydrolysis occurred immediately with most compounds; with others, after hours or days. A true ether sulfonate also gives an alkaline reaction, but the *p*H value is unchanged when it is mixed with water because hydrolysis cannot take place. In order not to disturb the natural acid reaction of the skin, it is advisable that cutaneous detergents should be active at a *p*H value corresponding to that of normal skin. Ether sulfonate may be adjusted to a *p*H of 5.5 by the use of a weak, nonirritating acid, and when such a solution is used for washing the skin or scalp, suds are produced which also have a *p*H value of 5.5. Therefore, when this detergent material is used on the skin, the surface of which also has a *p*H of 5.5, it is logical to assume that the normal acid balance will not be altered as it would be by the use of soap.

Most of the commercially available synthetic detergents are true sulfates and contain the hydrolyzable acid sulfate radical. Hence they release small quantities of sulfuric acid on standing or when used. This may be a contributing cause of the irritation reported as having been produced by these sulfated compounds[11]; for if a small amount of sulfuric acid, even in extreme dilution, is left on the skin, this nonvolatile acid becomes concentrated as the water evaporates. Moreover, the hydrolysis is accelerated in dilute acid solutions which, as stated,[7] are present on the skin.

Most if not all of these synthetic agents contain the sulfur joined directly to the oxygen atom and that, in turn, to a carbon atom, chemically represented as R-O-SO_2-OH. An ether sulfonate, however, contains the sulfur joined directly to a carbon atom, R-SO_2-OH. The latter compound cannot hydrolyze to produce sulfuric acid, as the bond R-S is extremely strong. This is not true of the weak bond R-O.

A SULFONATED ETHER DETERGENT CREAM

During a period of five years, in private practice and in the allergy and dermatologic clinics of the Massachusetts General Hospital, a detergent cream,[12] composed of a sulfonated ether, petrolatum, lactic acid and wool fat cholesterols, was used. This stable emulsion has the consistency of a thick dairy cream. It possesses active emulsifying, sudsing and dispersing properties. The surface activity is estimated to be about 40 per cent greater than that of soap.

Because its power to depress surface tension of water exceeds that of soap, this detergent cream is a more rapid and thorough cleanser of the skin, including the orifices of the pores and follicles, than soap is.

The cream spreads easily and requires little water to produce lather. Light friction aids emulsification of the resident oils and peptization

9. (*a*) McCarthy, L.: Diseases of the Hair, St. Louis, C. V. Mosby Company, 1940, p. 50. (*b*) Blank.[3a]

10. Sharlit, H.: Soap and the "Soap Problem," New York State J. Med. **43**:160 (Jan. 15) 1943.

11. (*a*) Biederman, J. B.: Sensitivity to Drene Shampoo, New England J. Med. **217**:1088 (Dec. 30) 1937. (*b*) Schwartz, L.: Actual Cause of Dermatitis Attributed to Socks, Pub. Health Rep. **49**:1176 (Oct. 5) 1934. (*c*) Osborne, E. D., and Putnam, E. D.: Industrial Dermatoses, J. A. M. A. **99**:972 (Sept. 17) 1932. (*d*) Blank.[3a] (*e*) McCarthy.[9a]

12. This product is manufactured by Fairchild Bros. & Foster, New York, under the trade name pHISODERM.

occurs, with subsequent dispersion of the soil into the suds. Rinsing is readily accomplished.

Because of the selective emulsifying power of the ether sulfonate and the emollient property of the preparation, the washing process does not dry the average skin or scalp. Some skins, however, secrete abnormally small amounts of sebum, and for this condition it was found advisable to prescribe the oily type of the cream, which contains about 50 per cent of emollient materials.

The cream itself, as well as the suds produced, has a *p*H value of approximately 5.5. It contains none of the fatty acids used in soaps and no perfume or coloring matter. It has been shown that (unlike soap, the sulfated oils or the alkyl sulfates) it may be left on the skin, when indicated, for a period of hours without harmful effects.

The detergent is active in hot or cold water, in soft or hard water and in sea water. Soap, of course, is precipitated by sea water, forming curds; few or no suds are formed, and the mixture is ineffective as a cleansing agent. This detergent, however, is not precipitated because compounds are formed which are soluble. Suds are produced, and effective cleansing is accomplished. It is therefore distinctly useful as a routine cutaneous detergent and a shampoo for maritime use. Its use should be valuable as adjunctive treatment of certain casualties encountered in naval warfare, such as immersion in oil. The unsuitability of soap for this purpose is well recognized.

The fact that the cream is a particularly active emulsifying agent for oils of all types explains its adaptability to the needs of industry. It is active under acid, neutral or alkaline conditions of use, and no insoluble soap residues remain on the skin or hair after rinsing.

Extensive use and results of cutaneous tests have proved that it is not a primary irritant and that it is hypoallergenic. Animal experimentation has proved that it is nontoxic and that it is less irritating to the eyes than soap.[13]

EXPERIENCE WITH THE DETERGENT

During a period of four years 374 patch tests with the detergent cream were included in a series of 8,628 tests with various allergens applied to several hundred patients with normal or abnormal skin. Positive vesicular reactions to this cream occurred in a fraction of 1 per cent. Approximately the same number of positive reactions to hydrous wool fat and to petrolatum was obtained. Another series of patients, 72, with asthma, hay fever or some form of allergic dermatitis were tested with this cream, my window patch test method being used.[14] The skin was observed for seventy-two hours, and all tests showed a negative response. The same patients were retested similarly after a two week interval, and negative results were again obtained. This experiment demonstrates that the product has low or no sensitizing properties.

The detergent has been used as a substitute for soap for various periods on over 2,000 persons with normal or abnormal skin. It has been used on the face, hands, scalp and body of infants, adults and aged persons. Some patients complained of a sensation of dryness after using the cream for the first time. This was considered to be due to the fact that they were accustomed to the feeling produced by the mildly keratolytic effect of alkaline soaps. This subjective feeling of dryness was relieved by the substitution of the oily type of detergents. When attempts were made to cleanse exuding, macerated or fissured surfaces, transient smarting sometimes occurred and occasionally aggravation resulted from the cleansing process. Approximately 200 patients have used this cream, excluding soap and all other detergents, for a period of from one to four years without having undesirable subjective or objective symptoms.

SUMMARY

Investigators in the field of dermatology and industrial medicine believe that the use of soap may initiate, aggravate or prolong certain types of dermatitis. Analysis of a survey of about 3,500 persons in diverse occupations disclosed that over 34 per cent believed that soap caused dryness, scaling, itching or burning or some combination of these symptoms. Because personal cleanliness is essential to good hygiene and social acceptability and because it usually is an adjunct of dermatologic therapy, it is necessary that a substitute for soap be made available.

In considering the properties of soap and a variety of substitutes for soap that have been used in recent years it is apparent that they do not fulfil all the qualifications of a desirable cutaneous detergent.

A detergent cream, composed of petrolatum, wool fat cholesterols, lactic acid and a sulfonated ether, has been used for five years in private practice and in clinics of the Massachusetts General Hospital. The detergent lowers surface tension of water considerably more than does

13. Tainter, M. L., and others: Unpublished data.

14. Guild, B. T.: Window Patch Test, Arch Dermat. & Syph. **39**:807 (May) 1939.

soap and is an active emulsifier of all types of oil. These properties make the cream about 40 per cent more effective than soap. It is more rapid in action than soap and requires less water and less friction to produce suds. It cannot hydrolyze to form sulfuric acid, as do the soluble alkyl sulfates. It contains no soap fatty acids and no perfume or coloring matter.

The cream, as well as the suds it generates, has a p_H value of 5.5, which corresponds to that of the average of normal skin. The cream is active under acid, alkaline and neutral conditions and when used with either hard or soft, cold or hot water. When used with sea water it is a good detergent and produces suds. When used with any of the aforementioned types of water, the detergent leaves no harmful residues on the skin or hair.

The preparation is no more toxic than soap would be if accidentally ingested. It is not a primary irritant. It is hypoallergenic.

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A SEVERE SYSTEMIC REACTION TO A BEE STING

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Reports on systemic reactions to bee stings are scarce, and the fact that a severe general reaction may develop in the absence of pronounced local inflammation has to my knowledge been emphasized only by Helm.[1] Thus the sequence and location of the cutaneous symptoms in the following report are noteworthy.

On an unusually warm and sunny day in January, while I was walking near the Beach Club of Santa Monica, Calif., in bathing trunks, without shoes, I stepped on one of many half-dead bees, lying in the sand near the water line. I felt a sharp pain on the plantar surface of the left great toe, which abated when I started to walk in the water. About ten minutes later there were tingling and stiffness of the palms, soon followed by numbness and edema of the lips, and by tingling over the scalp, in the axillas and about the genitalia. There was no pruritus at that time, but there was an urgent desire to touch the skin in the involved areas, and light stroking evoked a not unpleasant sensation. These symptoms were followed by generalized cutis anserina. About thirty minutes after the onset of the systemic reaction, malaise and abdominal cramps appeared; by that time angioneuritic edema of the eyelids and the lips was evident, and sharply demarcated, dusky erythema was present around the nasolabial folds, the perioral region and the chin. Within the next half-hour, urticaria, accompanied with intense edema of the prepuce, became manifest in the region covered by the bathing suit and later spread to the trunk. Pruritus was now intense; there was a feeling of tightness around the chest, and abdominal cramps were intensified. Only then did noticeable edema of the left great toe and the anterior part of the left foot develop. Inspection showed only a pin-sized puncture wound on the plantar surface of the toe. A subcutaneous injection of 1 cc. of a 1:1,000 aqueous solution of epinephrine hydrochloride, given at the nearest hospital, was followed within twenty minutes by almost complete relief. An hour or two later there was another attack of urticaria, with a recurrence of abdominal cramps and malaise. The symptoms were not severe, but they persisted for part of the following night. The next morning only moderate edema of the left great toe remained, which disappeared during the day.

From the department of dermatology of the University of Southern California.

1. Helm, S.: Severe Anaphylactic Reaction to Bee or Wasp Sting, Mil. Surgeon **92**:64, 1943.

Inquiry revealed that several members of the Beach Club had experienced general reactions to bee stings in the past; one man was said to have become unconscious a few minutes after being stung. It is supposed that the bees are blown on the beach periodically from nearby Catalina Island. A specimen submitted to the department of entomology of the University of Southern California was classified as apis mellifica, the common variety.

The rapid onset of the systemic reaction in my case and the loss of consciousness by the other member of the club are in accord with previously published reports. Helm,[1] von Geldern [2] and Thews [3] have emphasized the rapidity of onset: They have stated that urticaria, lacrimation, asthmatic attacks, localized and generalized edema, quickly ensuing unconsciousness and even a state of severe shock may occur. While there is agreement that individual hypersensitivity must exist, opinion as to the actual causal substance is divided. Benson and Semenov [4] have advanced the belief that sensitivity to protein in the stinger or the body of the bee is the responsible factor in most cases of severe systemic reaction. Gibb [5] has expressed the opinion that the patient may be reacting to pollen adhering to the insect and injected with the sting, a less likely theory.

Bee venom has not yet been successfully analyzed, although it is believed to resemble an albumin or to be of the nature of sapotoxin or cantharidin; consequently, its mode of action is subject to speculation. A logical suggestion has been advanced by Marcou and Derevici,[6] who

2. von Geldern, C. E.: Systemic Effects Following the Sting of Species of Epyris, Science **65**:302, 1927.

3. Thews, K.: Dangerous Allergic Reactions Following Bee Sting, Zentralbl. f. Chir. **63**:2497, 1936.

4. Benson, R. L., and Semenov, H.: Allergy in Its Relation to Bee Sting, J. Allergy **1**:105, 1930.

5. Gibb, D. F.: Anaphylaxis from Pollen Introduced by a Bee Sting, Canad. M. A. J. **19**:461, 1928.

6. Marcou, I.; Derevici, A., and Derevici, M.: Distribution of Histamine in Bee and Its Venom: Explanation of Cutaneous Reaction to Stings, Compt. rend. Soc. de biol. **126**:726, 1937.

found that the venom contains histamine, to the action of which the cutaneous symptoms are ascribed. Whatever the nature of the causative substance, the hypersensitivity can be successfully treated by injecting an extract made from the whole bee (Fisher [7]).

7. Fisher, D. C.: Hypersensitivity to Bees Successfully Treated with Whole Bee Extract, J. Allergy 5:519, 1934.

SUMMARY

A systemic reaction to a bee sting was observed and the occurrence of severe systemic reactions in the absence of pronounced local inflammation was noted. The remarkable sequence of distribution of the cutaneous manifestations—that is, palms, lips, scalp, genitalia and trunk—is thought to be of interest.

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LOW POTASSIUM DIET IN TREATMENT OF PSORIASIS

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During the past twenty-five years a great majority of the investigations relating to the cause and treatment of psoriasis have been based on the concept that the psoriatic lesions are a local cutaneous expression of a generalized and constitutional disturbance. The underlying disturbance has been variously considered as owing to faults in metabolism involving proteins,[1] carbohydrates [2] or fats [3]; to vitamin depletion, particularly vitamins C [4] and D,[5] and to endocrine disturbances related to the thymus,[6] thyroid,[7] pituitary,[8] gonads,[9] adrenals [10] or pancreas.[11]

The literature is both dogmatic and confusing. Evidence presented in support of each concept is for the most part balanced by reports expressing doubt or outright refutation. Striking "before and after" photographs are presented as visual proof of the benefits of a low nitrogen diet, a low fat diet or the use of adrenal cortex extract. Most of the procedures have failed to withstand general application because, in the main, only their protagonists have obtained satisfactory results or because the stringency of the therapeutic measures has made their continued use impractical. One observer, for example, reported favorable results from the administration of anterior pituitary extract, adrenal cortex extract and pancreatin and from the use of a low fat and a low protein diet, all being employed in the treatment of the same patient simultaneously.[12] In addition to the problem of making practical use of these various modes of treatment, the psychic factor, which is common to all forms of therapy in psoriasis, has made a critical estimate of their value difficult.

Two schools of thought as to the causation and therapeutics of psoriasis appear to have made much impression in the last decade. The theory of Gruetz and Buerger [3] involves a consideration of faulty fat metabolism and is based on their observation that there is a fasting hypercholesteremia and a disturbance in the fat tolerance curve in psoriasis and that a low fat diet produces amelioration of the psoriatic lesions. Conflicting reports [13] as to the hypercholesteremia have appeared both before and since the original article of Gruetz and Buerger. Moreover, Schaaf and Obtutowicz [14] and LeWinn and Zugerman [13k] were unable to demonstrate abnormal fat tolerance curves for patients with psoriasis. However, while there is some dissent,[13l] a majority of the subsequent reports by other authors are in

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1. (*a*) Schamberg, J. F.; Kolmer, J. A.; Ringer, A. I., and Raiziss, G. W.: J. Cutan. Dis. **31**:802, 1913. (*b*) Schamberg, J. F.: Dietary Treatment of Psoriasis, J. A. M. A. **98**:1633 (May 7) 1932.

2. Lortat-Jakob, Legrain and Pellissier: Bull. Soc. franç. de dermat. et syph. **33**:101, 1926.

3. Gruetz, O., and Buerger, M.: Klin. Wchnschr. **12**:373, 1933.

4. Lutz, W.: Schweiz. med. Wchnschr. **65**:1169, 1935.

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support of the benefits derived from a low fat diet.[15] The more recent work with soybean lecithin[16] deals with lipids instead of total fats. The results so far appear to parallel those obtained with a low fat diet.

The second group of investigations began with Grueneberg's[10] use of adrenal cortex extract in the treatment of psoriasis. There have been reported three fairly well established facts which could be interpreted as being indicative of a disordered adrenal cortex metabolism in psoriasis: (1) a definite and decided diminution in urinary vitamin C excretion,[17] (2) a decrease in the amount of sulfur excreted in the urine but which may be augmented by feeding ascorbic acid,[17a] (3) an increase in the blood potassium level.[18] From these facts may be inferred a relationship between psoriasis and the adrenal cortex, as follows:

1. Vitamin C appears to be intimately associated with the function of the adrenal cortex. Goldzieher[19] has recently summarized this relationship. The concentration of ascorbic acid in the adrenal cortex is greater than that in any other tissue. The hemorrhages and degenerative changes which develop in the adrenal cortex in the course of scurvy have been shown to be directly related to the vitamin C deficiency. In states of adrenal cortex insufficiency, such as postadrenalectomy, Addison's disease and experimental intoxication with diphtheria toxin, injections of adrenal cortex extract are more effective if combined with the administration of ascorbic acid.

2. The adrenal cortex not only is rich in sulfur but appears to have a major role in sulfur metabolism.[19]

3. The pronounced loss of sodium and the retention of potassium in adrenal cortex insufficiency are well known. Recent reports indicate that adrenal cortex control of the plasma potassium level is of major importance, with the sodium content of the plasma a secondary factor.[19]

Shortly after Gruetz and Buerger introduced a low fat diet for the treatment of psoriasis, Grueneberg[10] began to publish a series of reports concerning the therapeutic value of adrenal cortex extract for this disease. By 1938, after five years' experience with 250 patients, Grueneberg[20] expressed his conviction that adrenal cortex extract is distinctly effective in the treatment of psoriasis. However, in the hands of other observers, this form of therapy has not produced uniformly successful results. Reports have been favorable,[21] unfavorable[22] or equivocal.[23]

The use of adrenal cortex extract is to some degree limited by the rather high cost of this material. In order to modify this difficulty, as well as to study the physiologic considerations involved, Incedayi and Ottenstein[21c] enhanced the action of the injected adrenal cortex extract by means of a low potassium diet. They reported striking improvement in patients treated by this method.

In 1935 Lutz[4] reported definite, although temporary, amelioration of the disease in 3 patients with severe psoriasis after administration of ascorbic acid in large doses. Although similarly good results were noted by Volpe,[24] others[25] have failed to observe distinct benefits.

Incedayi and Ottenstein[17c] also employed ascorbic acid in the treatment of psoriasis. However, they simultaneously prescribed a low potassium diet, attempting by this means to modify the action of the adrenal cortex. The good results obtained with this combined therapy led them to state that the unfavorable responses to administration of ascorbic acid alone noted by other observers were due to failure to modify adrenal cortex function. They expressed their belief that even better results would be obtained

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in the treatment of psoriasis with large doses of ascorbic acid, a low potassium diet and injections of adrenal cortex extract employed simultaneously. Since no subsequent report of such a treatment has appeared in the available literature, we have studied a group of patients with psoriasis, in accordance with this procedure.

MATERIAL AND METHODS

Subjects.—Eighteen patients were selected for treatment on the basis of an unquestionable diagnosis of psoriasis, duration of symptoms, previous therapy and response, and willingness of the patient to cooperate closely. In all patients the lesions were well established and in most instances widely distributed. The activity of the latter was mainly acute or subacute. Mildly active arthropathy was present in 2 patients.

Procedure.—The studies were begun, as far as possible, in November and December 1943 and January 1944 in order to permit full seasonal establishment of the lesions and to obviate the question of seasonal improvement with the advent of summer. All patients were ambulant, and in no case was hospital management instituted. Therapy was begun with the oral administration of 300 mg. of ascorbic acid per day for one week, after which, in addition, intramuscular injections of adrenal cortex extract (Upjohn) were given in doses of 25 dog units twice weekly. After three to four weeks of this regimen, a low potassium diet was begun. While this was not a weighed diet, the relative size of portions of the various foods was discussed with the patients, as in the management of diabetes mellitus. In order to simplify the diet to assure a daily intake of potassium as close as possible to the optimum of 1.6 Gm., all foods with a potassium content greater than 0.30 per cent were prohibited (see table). In addition, the patients were encouraged to increase their intake of sodium chloride. In most instances, the ascorbic acid dosage was ultimately increased to 600 mg. per day, while the amount of adrenal cortex extract was raised to 50 dog units twice weekly. Patients refrained from other forms of therapy. For the relief of itching and dryness of the lesions, an ointment of equal parts of hydrous wool fat and petrolatum was prescribed.

RESULTS

There was no instance of maintained improvement in any of our 18 patients. One patient was forced to discontinue treatment after the fifth week because of distressing exacerbations following each dose of adrenal cortex extract. In most patients there was no alteration in the lesions other than the ordinary fluctuations in acuity seen in the course of psoriasis. In patients with arthropathy, no benefits were found as a result of treatment. Photographs were taken in many cases before the inception of treatment. We were unable to note any net change in the severity or extent of the lesions at the time treatment was discontinued.

The most promising result was observed in the case of a 37 year old man, whose psoriatic lesions first appeared at the age of 7 years. In this patient, as in the others, the preliminary week of ascorbic acid therapy produced no change. However, after the first two doses of adrenal cortex extract, the small lesions showed distinct blanching at the periphery, while the larger, confluent lesions exhibited much less redness than before and a decrease in the thickening. After the fourth dose all the lesions appeared definitely paler, the texture of the skin was softer and the patient reported a lessening of the itching. After the

*Percentage of Potassium Content of Various Foods**

Food	Percentage of Potassium
Meat extracts	4.16
Lima beans	1.701
Wheat bran	1.217
Prunes, dried	1.004
Potato chips	0.951
Raisins	0.859
Nuts, all kinds	0.58
Spinach, uncooked	0.507
Dates	0.504
Broccoli, edible portion	0.468
Sweet potato	0.397
Oatmeal	0.396
Bananas	0.391
Endive	0.389
Beef (20% protein)	0.368
Lettuce	0.356
Celery	0.292
Pineapple	0.270
Tomatoes, or juice	0.267
Egg plant	0.259
Radishes	0.256
Peaches, fresh	0.214
Currants, fresh	0.211
Bread, whole wheat	0.206
Spinach, boiled and drained	0.200
Grapes	0.197
Onions, uncooked	0.182
Oranges, or juice	0.181
Cottage cheese	0.177
Raspberries, fresh	0.171
Ice cream, commercial	0.167
Bacon or pork	0.164
Blackberries	0.160
Grapefruit	0.161
Buttermilk	0.153
Strawberries	0.150
Squash, summer	0.150
Oysters, large	0.143
Milk, whole	0.141
Cucumbers	0.140
Eggs	0.138
Pears, fresh	0.136
Cornflakes	0.134
Macaroni	0.130
Cream, average	0.130
Apples, fresh	0.127
Jelly made with fruit juice	0.118
Parsnips, boiled and drained	0.107
Cabbage, boiled and drained	0.103
Peas, boiled and drained	0.103
Pineapple, canned	0.103
Beans, string, boiled and drained	0.101
Cauliflower, boiled and drained	0.101
Peaches, canned	0.101
Raspberries, canned	0.101
Lemons	0.101
Watermelon	0.101
Farina	0.101
Sweet corn	0.101
White bread	0.098
Coffee, beverage	0.100
Oysters, small, raw	0.096
Cheese, hard	0.080
Turnips, boiled and drained	0.075
Squash, winter, boiled and drained	0.073
Pumpkin, boiled and drained	0.073
Carrots, boiled and drained	0.072
Asparagus, boiled and drained	0.070
Onions, boiled and drained	0.070
Pears, canned	0.070
Rice, white, raw	0.070
Huckleberries or blueberries	0.061
Margarine	0.048
Farina, cooked	0.040
Tapioca	0.039
Tea, beverage	0.030
Butter	0.014
Mayonnaise	0.007
Sugar, white	0.004

* From Goldzieher, M. A.: The Adrenal Glands in Health and Disease, Philadelphia, F. A. Davis Company, 1944, p. 400.

daily dose, while a few new lesions appeared on the face and neck at the hair line, the patient stated that he was able to go as long as twenty-four hours without application of the palliative ointment, whereas the itching and burning had previously made application necessary at least every twelve hours. At this time, seventeen days after beginning therapy with adrenal cortex extract, the patient had received a total of 175 units of cortex extract and 7,200 mg. of ascorbic acid. There was notable blanching of all major areas with decided thinning of the centers and exfoliation of the scales. During the next two weeks progressive improvement was noted in all areas except the scalp, which continued to itch and scale as actively as before therapy was begun. In the sixth and seventh weeks no improvement ceased and many small new lesions began to appear. By this time, a total of 350 units of adrenal cortex extract had been administered and the patient had taken 16,800 mg. of ascorbic acid. At this point the daily dose of ascorbic acid was increased to 600 mg., and the semiweekly dose of adrenal cortex extract was raised to 50 units. For the next two weeks there was no change in the lesions. At the beginning of the tenth week the patient was placed on a low potassium diet, while the daily dose of ascorbic acid was maintained at 600 mg. and 50 units of adrenal cortex extract were given twice weekly. Within four days there was striking improvement in all areas, including the scalp. There was little change during the next five weeks, the lesions showing exacerbations and remissions as ordinarily seen in psoriasis treated by other means. In the sixteenth week many new lesions began to appear, the older lesions showed unquestionable progressive exacerbation and treatment was discontinued. An optimistic statement of the patient's condition as the result of therapy with ascorbic acid, adrenal cortex extract and a low potassium diet would be "moderately improved."

In connection with this case report it is interesting to note that the patient had previously been treated with roentgen ray irradiation, ultraviolet irradiation, various ointments (including chaulmoogra oil), intravenous injections of thiamine hydrochloride and a low fat diet. All procedures had been strikingly effective at first but failed to maintain improvement.

COMMENT

A review of the literature concerning investigations of psoriasis reveals little which can be accepted as factual. Agreement between the reports which even remotely approach unanimity can be found for the most part in relation to sulfur metabolism and vitamin C metabolism. In their publications Grueneberg [19] and Klauder and Brown [26] are in agreement that the skin of psoriatic patients contains abnormally large amounts of sulfur. As already noted, a fairly uniform concurrence exists that there is an apparent deficiency in vitamin C excretion in the urine of patients suffering from psoriasis. The work of Reiss [27] reveals a relationship between these two substances in that there is a decreased excretion of both in the urine in psoriasis and that increased vitamin C intake by any route augments not only the excretion of urinary vitamin C but that of urinary sulfur. However, since there is no conclusive evidence that vitamin C therapy, and therefore increased sulfur excretion, ameliorates psoriasis, there is some doubt whether disturbed sulfur metabolism is directly concerned in the causation of psoriasis.

It seems to us that the implication of disturbed vitamin C metabolism as either a specific effect or a cause of psoriasis is fallacious. One of the best recognized functions of vitamin C is its role in oxidation-reduction processes. With the heightened local metabolism in the inflamed psoriatic lesions, it is only reasonable to expect increased utilization and decreased urinary excretion of vitamin C.

It is worthy of note that none of the characteristics of adrenal cortex insufficiency have been described as part of the symptoms of psoriasis. Assuming that minimal adrenal cortex hypofunction exists in psoriasis, the dosage of adrenal cortex extract employed by us, as well as by other observers, should be at least uniformly curative. Instead, there is considerable disagreement as to results obtained. The combined use of adrenal cortex extract, high vitamin C intake and a low potassium diet is a procedure which under ordinary circumstances should be adequate to control frank, although mild, Addison's disease. Using these methods of treatment simultaneously, we were unable to influence the course of psoriasis. We, therefore, do not feel that there is justification for the conclusion that a disturbance in adrenal cortex function exists in this disease.

SUMMARY

Eighteen patients with psoriasis were treated with ascorbic acid, adrenal cortex extract and a low potassium diet. We have been unable to observe any definite benefit from this form of therapy.

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CUTANEOUS REACTIONS OF PERSONS WITH ATOPIC ECZEMA TO HUMAN DANDER

RESULTS OF PATCH TESTS ON SCARIFIED SKIN

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The presence of allergenic activity in human dander has been reported by Van Leeuwen and his co-workers,[1] Keller[2] and Hampton and Cooke[3] and has been discussed several times by Sulzberger. Most allergists, however, have expressed doubt that human dander is allergenic for man.

Human dander is a complex mixture of cornified epidermal cells, sebum, sweat and numerous micro-organisms, as well as various dust particles and other material. In recent studies I[4] presented evidence confirming the existence of an allergen for man in human dander. The allergen investigated was found to be separate and distinct from the allergens in feathers, wool, orris, house dust, cottonseed, karaya gum, bluegrass and ragweed pollen. It was found in dander and hair from the scalp but was not detected in scales of skin from the general body surface or in hair, other contents or inner lining of a dermoid cyst of the ovary. The biologic origin of this allergen has not been determined. Its presence was demonstrated by means of cutaneous reactions in certain patients with atopic eczema. In 9 of 11 adults with this disease urticarial reactions to scratch tests were obtained, and reagins were demonstrated by local passive transfer.

Because of the reports of Peck and Salomon[5] and Albert and Walzer[6] and the discussions of Sulzberger[7] on transepidermal penetration of protein or protein-like allergens and because of clinical considerations of exposure to dander, it was deemed desirable to perform patch tests. Results of these were positive in 15 of 20 children with eczema. Typical eczematous lesions were reproduced by rubbing human dander on uninvolved areas of the abdomen or back.[4c] In the case of the 9 adults with urticarial reactions to and reagins for human dander, referred to in the preceding paragraph, patch tests were negative. These required further study, a report of which is presented.

EXPERIMENTAL STUDIES

Dander (dandruff) was combed from the heads of persons with normal scalps, separated from hair, extracted with ether and dried. It was then extracted with 2 volumes of 50 per cent glycerin, and the resulting extract was passed through a Seitz filter. This extract was used for scratch tests in 1:10 and 1:100 dilutions. (The undiluted extract preparation was found to be irritating to all skins tested, including those of persons without atopic eczema.)

For the patch tests human dander was mixed with sufficient petrolatum to make a thick paste (1 part of dander to 1 part of petrolatum). This was spread on a piece of white blotting paper and applied with adhesive tape directly to the skin. But for the exceptions noted later, patches were allowed to remain in contact with the skin for two days. Reactions were read at the time of the removal of the patch, one half hour, one day and seven days later.

Ten patients with atopic eczema were studied. Data for 4 of these persons were included in the first report

From the Departments of Medicine and of Bacteriology and Immunology of the University of Louisville School of Medicine.

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2. Keller, P.: Relations Between Asthma and Eczema, Arch. f. Dermat. u. Syph. **148**:82, 1924.

3. Hampton, S. F., and Cooke, R. A.: The Sensitivity of Man to Human Dander with Particular Reference to Eczema (Allergic Dermatitis), J. Allergy **13**:63 (Nov.) 1941.

4. Simon, F. A.: (*a*) On the Allergen in Human Dander, J. Allergy **15**:338 (Sept.) 1944; (*b*) Skin Reactions to Patch Tests with Human Dander, Ann. Allergy **2**:109 (March) 1944; (*c*) Human Dander: An Important Cause of Infantile Eczema, J. A. M. A. **125**: 350 (June 3) 1944.

5. Peck, S. M., and Salomon, G.: Eczema of Infancy and Childhood, Am. J. Dis. Child. **46**:1308 (Dec.) 1933.

6. Albert, M., and Walzer, M.: (*a*) Contact-Reactions in Atopy: Contact Reactions to Silkworm in Atopic Subjects, J. Immunol. **38**:201 (March) 1940; (*b*) Contact Reactions in Atopy: Incidence of Contact Reactions with Various Allergens, J. Invest. Dermat. **3**: 119 (April) 1940; (*c*) Contact Reactions in Atopy: Contact Reactions in Various Atopic Illnesses, J. Allergy **14**: 437 (Sept.) 1943.

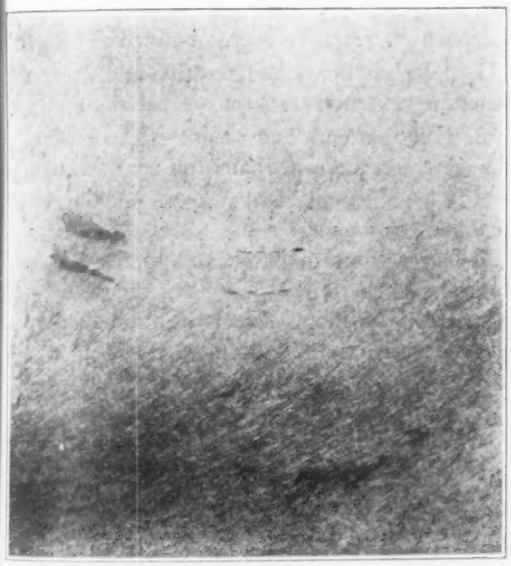
7. Sulzberger, M. B., in discussion on Albert and Walzer.[6b]

this subject.[4a] Six were new patients, who had typical lesions on the face, neck and shoulders, in the antecubital spaces and on the forearms. In 2 patients the scalp was involved. In most of these persons the popliteal regions had been involved earlier in life. Seven patients had suffered with the disease since infancy. All have family histories which were positive for atopy.

Cutaneous Reactions to Human Dander in Patients with Atopic Eczema

Patient No.	Age, Yr.	Urticarial Reaction to Scratch Test Dilutions		Eczematous Reaction to		
		1:10	1:100	Patch Test on Normal Skin	Patch on Scratch Test	Massage Test
1	21	++	+	—	++	—
2	29	++	±	—	+++	—
3	17	++	+	—	—	—
4	30	—	—	—	++	—
5	18	++	±	—	+	—
6	14	—	—	± (+)	+	+
7	27	—	—	++	+++	+
8	44	+	—	—	++	—
9	21	++	+	+	++	+
10	42	++	+	—	++	—

Routine cutaneous tests elicited urticarial reactions to foods or inhalants or both in 9 of the 10 patients. Six patients had asthma or hay fever or both, in addition



Cutaneous reaction to a patch on scratch test. On the left are shown two superficial scratches which were made on the skin and to which human dander was applied as a patch test. The patch remained in position for two days. The photograph, taken two days after removal of the patch, shows two linear crusts formed at the sites of two shallow, superficial vesicles surmounted on areas of slight redness and swelling. On the right, barely visible, are two scratches of the same depth, made at the same time, to which a blank piece of blotting paper had been applied.

to atopic eczema. In the case of the infants and children in whom reactions to patch tests were positive, the allergen evidently reaches the sensitive tissue by direct penetration through the cornified layer of the epidermis, or through hair follicles or through gland ducts. In the adults, however, in whom results of patch tests were negative (note exceptions mentioned subsequently and shown in the accompanying table) the question arises: If human dander is really an allergenic excitant of atopic eczema in these patients, by what route does the allergen reach the sensitive tissue? An indirect route by way of the blood stream seemed improbable; hence, further attention was turned toward direct applications to the skin surface.

Patch tests were applied to areas of normal skin of the back and allowed to remain in position for two days. Positive reactions were obtained in 2 of the 10 patients (table, patients 7 and 9). By allowing patches to remain in position for seven days 1 additional positive reaction was obtained (patient 6). Mixtures of dander in petrolatum and also in soap solution were massaged into the skin with the end of a blunt wooden applicator. In 3 patients positive reactions resulted. These consisted of papules located at the sites of hair follicles. A third type of test was then applied, namely, a patch on scratch test. Three scratches about 5 mm. in length and 1 to 2 mm. apart were made in the skin of the back. Bleeding did not occur. A patch was applied immediately on these scratches and allowed to remain in contact with them for two days.

In 9 of the 10 patients eczematous reactions developed at the site of the test. These consisted of papules, vesicopapules, oozing of serous fluid, crust formation, redness and swelling. The papules were located in and immediately adjacent to the scratches. The redness and swelling extended 1 to 3 mm. beyond them. The milder reactions consisted in redness and swelling alone, without papules. Desquamation occurred in one to two weeks. The reactions remained visible for one to four weeks. In 10 persons without eczema all of these tests elicited negative reactions.[8]

COMMENT

The occurrence of reactions to human dander applied to previously scratched skin of certain patients with atopic eczema in whom patch tests on uninjured skin produce no reactions is probably due to the fact that the scratches remove the barrier of cornified epidermis. Thus the allergen is permitted to penetrate to the sensitive tissue. In a small percentage of adults and a larger percentage of children [4c] with the disease, the cornified layer of epidermis does not prevent penetra-

8. In several instances a very slight erythematous reaction (hardly perceptible) developed at sites of scratches to which the dander had been applied, while no reaction developed at sites of scratches to which a blank patch had been applied. A similar very slight erythema has been observed in several normal, noneczematous children at sites to which dander had been applied to normal, uninjured skin. There are two explanations for these reactions: (1) that they are a result of a primary irritant which manifests itself not only in the urticarial reactions to scratch and intradermal tests but in the eczematous reactions to patch tests (including patch on scratch tests); (2) that they are manifestations of a slight, subclinical hypersensitiveness to an allergen in human dander.

tion of the allergen in reactive concentrations. In these persons reactions may be obtained to patch tests on uninjured skin.

The reactions to patch tests, including patch on scratch tests, resemble closely, in their general appearance, development and retrogression, the naturally occurring lesions of the disease. Patients with atopic eczema, like all other persons, are exposed to human dander from their own scalps, from the scalps of other persons or from both.

The concept of human dander as an important allergenic excitant of atopic eczema will account for the following clinical characteristics of the disease: (1) its occurrence in infants who are receiving nothing by mouth except human milk, (2) the frequent failure of dietary manipulation to result in healing of the lesions, even in patients whose diets are based on the elimination of foods which give positive urticarial reactions to scratch or intradermal tests, (3) the tendency of the disease to lessen when the patient is in the hospital, (4) the predilection of the lesions for cutaneous areas near the scalp and in infants, for areas not covered by clothing, (5) perhaps even the propensity of the lesions to occur on flexural surfaces, such as the cubital space, which may be areas permitting greater penetration of allergen, and (6) perhaps also the tendency of the disease to heal and disappear in hot weather, for it appears to be a clinical fact that in hot weather not only is there less dander on the scalp but that which is present is less dry and less easily scattered about. The less frequent involvement of the scalp as compared with other areas must be explained on the assumption that the threshold of reactivity of the scalp is greater than that of other skin areas. Incomplete experiments now in progress indicate that this is actually the case.

The existence of other important causative factors (possibly allergenic excitants?) in addition to human dander is indicated by observation of the following characteristics. In certain patients the massive intense exposure of a given cutaneous area to human dander, produced by a patch test (on normal skin or on skin which has previously been scratched), results in an eczematous reaction of slight or moderate intensity, while on an area only a few centimeters distant a more intense, naturally occurring, eczematous reaction exists. It is illogical in such instances to ascribe the latter eczematous reaction entirely to casual contact with human dander. Nor will the assumption of localized variations in specific skin sensitivity account for the observation, because experiments now in progress indicate that various cutaneous areas of the body (with the exception of the scalp) exhibit relatively minor variations in their eczematous response to human dander.

SUMMARY

Ten patients with atopic eczema were tested with human dander. Seven patients had positive and 3 negative urticarial reactions to scratch tests. Eczematous reactions were obtained as follows:

1. Three patients had positive and 7 negative reactions to patch tests on normal skin.

2. Nine patients had positive reactions and 1 a negative reaction to patch on scratch tests.

3. Three patients had positive and 7 negative reactions to massage tests.

The reactions to patch and to massage tests reproduced the typical lesions of the disease. In 10 persons without eczema results of all tests were negative.

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Abstracts from Current Literature

EDITED BY DR. HERBERT RATTNER

STUDIES ON PALMAR SWEATING. JACOB J. SILVERMAN and VERNON E. POWELL, Am. J. M. Sc. **208**:297 (Sept.) 1944.

Silverman and Powell describe an inexpensive and relatively simple technic for study of palmar sweating, especially that which results from emotional, intellectual and sensory stimuli. They point out that palmar sweating is continuous, is practically uninfluenced by outside temperatures and may be evoked by mental stimuli. The palm is one of the few places where emotional sweating takes place, and palmar sweating peculiarly is an indicator of emotional disturbances. The authors report a study of the palmar sweat response of more than 1,100 patients in an army general hospital. Approximately 25 per cent of the patients showed an intense response (as recorded by their technic). In those patients there invariably was evidence of coincidental emotional strain or disturbance of the autonomic nervous system.

THE EFFECT OF CALCIUM PANTOTHENATE AND PARAAMINOBENZOIC ACID ON GRAY HAIR IN MAN: A STUDY ON A GROUP OF YOUNG AND OLDER INDIVIDUALS. HAROLD BRANDALEONE, ELIZABETH MAIN and J. MURRAY STEELE, Am. J. M. Sc. **208**:315 (Sept.) 1944.

Brandaleone, Main and Steele report that of 19 elderly persons with gray hair and 14 younger persons, only 2 exhibited a significant change in color of hair after a six to eight month period of administration of large amounts of calcium pantothenate and concentrated yeast. All but 6 were also given paraaminobenzoic acid.

LYNCH, St. Paul.

REVIEW OF 2,144 COURSES OF RAPID TREATMENT FOR EARLY SYPHILIS. E. W. THOMAS and GERTRUDE WEXLER, Am. J. Syph., Gonor. & Ven. Dis. **28**:529 (Sept.) 1944.

The authors started the rapid treatment of early syphilis at Bellevue Hospital in December 1939. The most effective method with mapharsen alone during a period of from five to ten days requires a total dose of over 1 Gm. When it was given in amounts which appreciably exceeded 1 mg. per kilogram of body weight on any one day the incidence of arsenical encephalopathy was more than 1 per cent. In the administration of 909 such courses of treatment, each lasting from six to ten days, 3 deaths occurred.

In a series of 1,046 courses of treatment, each consisting of daily injections of mapharsen in doses of about 1 mg. per kilogram of body weight and four fevers induced by typhoid vaccine during a ten day period, there were only 3 cases of mild arsenical encephalopathy and no deaths. In a total of 2,144 courses of rapid treatment the incidence of encephalopathy was higher among men than among women.

Results of quantitative serologic tests during and after the administration of rapid treatment suggest that reagin may be produced for varying lengths of time after the spirochetes have been eradicated. In general, the longer a patient has had syphilis the longer it is before the serologic reaction for syphilis becomes negative.

Of all patients followed for six months or more after treatment, 80 per cent demonstrated results that were considered to be satisfactory. If patients who were retreated are included 85.6 per cent had results that were considered to be satisfactory. The low incidence of positive spinal fluid findings from six months to four years after rapid treatment is encouraging. Neurorecurrences after rapid treatment have been fewer than after routine treatment.

A few patients were treated according to a plan of therapy devised by Eagle, in which three injections of mapharsen were given each week during a period of from six to eight weeks. Difficulty was experienced in persuading patients to complete the course of treatment and to report regularly; hence the number of patients treated was too small for evaluation.

INTENSIVE CHEMOTHERAPY OF EARLY SYPHILIS. A. W. NEILSON, L. F. BLANEY, L. J. STEPHENS and R. W. MAXWELL, Am. J. Syph., Gonor. & Ven. Dis. **28**: 553 (Sept.) 1944.

Four hundred and eighty-seven patients with early syphilis were treated intensively with mapharsen, each receiving from 900 to 1,200 mg. during an average period of five days. More than one half of the patients were treated additionally with an insoluble bismuth preparation in an attempt to create a chemotherapeutic regimen of maximum effectiveness. Four fatalities are discussed in detail. The fatality rate of patients treated was 0.76 per cent.

The Kahn reaction became negative after an average of four and three-tenths months in patients who received mapharsen alone and after an average of two and eight-tenths months in patients who received bismuth in addition to mapharsen. Fifteen per cent of all patients observed for five months or longer experienced serologic or mucocutaneous relapse.

Twenty pregnant women were given therapy, 17 of whom were kept under observation until delivery had occurred. Only 2 infants exhibited definite indications of having congenital syphilis and 2 (under 3 months of age) had positive reactions to the Kahn test.

It is the authors' opinion that the rate of complete cure in this group of patients will not be less than 70 per cent.

SYPHILIS IN GONORRHEA PATIENTS AND CONTACTS. N. W. GUTHRIE, Am. J. Syph., Gonor. & Ven. Dis. **28**:583 (Sept.) 1944.

Thirty-one per cent of patients with gonorrhea and 39 per cent of their contacts had or had had syphilis on admission to Johns Hopkins Hospital during a twenty-two month period. One hundred and twenty-eight nonsyphilitic patients with gonorrhea were reexamined for the presence of syphilis during the twelve weeks after admission. Three were observed to have contracted the disease. This relatively low rate at which syphilis develops in patients with gonorrhea does not justify frequent testing of the blood. Patients should

be inspected for the presence of lesions when the necessary observations for gonorrhea are made. Serologic tests for syphilis should be made on their admission to the hospital and at four to six week intervals during the next twelve weeks.

REUTER, Milwaukee.

ECZEMA—ALLERGIC DERMATITIS. STEPHAN EPSTEIN, Ann. Allergy **2**:247 (May-June) 1944.

In a comprehensive review of recent literature regarding eczema and allergic dermatitis, Epstein cites 123 references. The article should be read in its entirety by every one interested in this subject.

Especially important appear to be the contributions on autosensitization, on sensitivity to human dander, on industrial dermatitis and on the prospect of a nonspecific-specific therapy for atopic dermatitis and perhaps other forms of eczema by the conjugated histamine antigen (hapamine).

HISTOPATHOLOGY OF ECZEMATOID DERMATOSES. WILBERT SACHS, CHARLES S. MILLER and MARGARET GRAY, Ann. Allergy **2**:289 (July-Aug.) 1944.

In a histologic study of four types of the eczematoid dermatoses the authors draw the following conclusions:

"1. Contact dermatitis differs from the other diseases by the type of vesicle, little or no acanthosis, and a mild superficial inflammatory reaction.

"2. Neurodermatitis has a nonedematous regular acanthosis, thickening of the walls of the small arteries and a focal cellular reaction.

"3. Nummular eczema has the epidermis and cutis of neurodermatitis, plus an intraepidermic vesicle.

"4. Eczema is not a disease *sui generis* but probably an expression of several diseases having similar findings. It differs from the other three by an extensive cutis reaction involving the capillaries and intense diffuse cellular reactions. There is also an irregular acanthosis, most often with all signs of edema and sometimes with little or none."

SHAW, Chattanooga, Tenn.

CONTACT DERMATITIS FROM RUBBER GAS MASK. JOE C. GILBERT, Ann. Allergy **2**:339 (July-Aug.) 1944.

Gilbert reports another instance of dermatitis resulting from use of a gas mask. A patch test with the gas mask rubber elicited a positive reaction. A different type of gas mask was suggested for the patient.

PERLÈCHE—ITS NOSOLOGIC STATUS. CLARK W. FINNERUD, J. A. M. A. **126**:737 (Nov. 18) 1944.

Perlèche should be regarded no longer as a disease entity but rather as an abnormal cutaneous change occurring in the form of an intertrigo of the labial commissures. Probably, therefore, this term should be appropriately modified in use in order to suggest in a specific instance the cause or the nature of the condition which it names: Infectious perlèche could apply to the condition when it is considered to be primarily of bacterial or mycotic origin. Malocclusion, or mechanical perlèche, might signify the condition (as seen in elderly persons) when it is caused by a mechanical agent, such as ill fitting dentures. Vitamin deficiency perlèche could specify the condition when it is of hypovitaminotic or avitaminotic origin. Idiopathic perlèche could designate the condition in instances when no causative factors can be determined.

HENSCHEL, Denver.

THE INITIATING AND PROMOTING ELEMENTS IN TUMOR PRODUCTION: AN ANALYSIS OF THE EFFECTS OF TAR, BENZPYRENE AND METHYLCHOLANTHRENE ON RABBIT SKIN. WILLIAM F. FRIEDEWALD and PEYTON ROUS, J. Exper. Med. **80**:101 (Aug.) 1944.

Previous studies of the effect of tar on rabbit skin have demonstrated that the carcinogen acts in two ways. First, it changes normal cells into neoplastic cells, and second, it encourages the multiplication of the latter. Certain other agents cannot initiate neoplastic change but they can stimulate proliferation of normal epidermal cells and can promote the development of hidden entities which otherwise never would have been of any significance. The present studies with rabbits demonstrate that benzpyrene rapidly induces neoplastic changes but has only slight power to encourage multiplication of the neoplastic cells. Methylcholanthrene has somewhat more and tar considerably more of the latter ability. The character of the benign tumors is about the same in each case; but the pure hydrocarbons excite the connective tissues only slightly and furnish a scanty stroma, and the tumors remain small and indolent. Thus, in appraising the action of carcinogens, one must take into account not only the capacity of these agents to induce neoplastic change and to promote, or perhaps suppress, tumor growth but the ability to condition to no inconsiderable extent both the type and the structure of the developing tumor.

LYNCH, St. Paul.

A NEW METHOD OF GIVING POTASSIUM IODIDE. W. T. GARFIELD, New England J. Med. **229**:971, 1943.

Garfield describes the use of potassium iodide administered in the form of enteric-coated pills, each containing 1 Gm. (15 grains) of the drug, in 12 cases of syphilis. This form of treatment is recommended because it does not precipitate gastric disturbances and because it admits of accuracy in determining the dose.

TROPICAL DISEASES OF THE SKIN. H. FOX, New England J. Med. **231**:482 (Oct. 5) 1944.

Five tropical diseases (yaws, pinta, verruga peruana, cutaneous leishmaniasis and leprosy) are discussed with the purpose of emphasizing their increasing importance (1) to physicians whose practice may include returned veterans who have contracted a tropical disease while in military service, and (2) to medical officers whose responsibilities may include care of native populations in tropical areas.

RONCHESE, Providence, R. I.

PENICILLIN IN THE TREATMENT OF EXPERIMENTAL INFECTION WITH BACILLUS ANTHRACIS. F. R. HEILMAN and W. E. HERRELL, Proc. Staff Meet., Mayo Clin. **19**:492 (Oct. 4) 1944.

Fifty-five per cent of mice treated with penicillin were protected against 10,000 times the lethal dose of Bacillus anthracis, even when sixteen hours had elapsed between the inoculation and the beginning of treatment. It is evident that infections with B. anthracis are susceptible to treatment with penicillin.

HENSCHEL, Denver.

BRIEF HISTORY OF THE SCHOOL OF ST-LOUIS. ALBERIC MARIN, Union méd. du Canada **73**:1285 (Nov.) 1944.

The history of dermatology and syphilology in France is intimately connected with that of l'hôpital St-Louis in Paris. It is true that other centers, such as those at Montpellier, Toulouse, Bordeaux, Lyon and Stras-

burg, became famous; but it was the work produced by the Paris school that was most remarkable.

L'hôpital St-Louis, founded by Henry IV in 1607, was used at first for patients with plague or other contagious diseases; on certain occasions patients with tuberculosis were admitted. At that time the hospital functioned only intermittently during epidemics. When conditions of health improved, the hospital was closed. There were always more patients than beds; for example, in 1790 four hundred and fifty-three beds contained 700 patients.

The modern period of dermatology began in 1801 with Alibert, who interested himself immediately in diseases of the skin, about which little was known. In 1804 he began his celebrated lectures, which attracted many students and physicians. Alibert was the first professor of dermatology in Paris and established the universal renown of l'hôpital St-Louis for the study and treatment of dermatoses.

Later Biett first described the epidermic collaret of papular syphilids; Cazenave studied lupus erythematosus and pemphigus foliaceus; Gibert individualized pityriasis rosea; Devergie first described pityriasis rubra pilaris; Fazin isolated fungi and discussed mycosis fungoides and erythema induratum; Hardy simplified the treatment of scabies; Vidal proved that pemphigus neonatorum is contagious; Besnier popularized the use of biopsy; Hallopeau described acrodermatitis continua; Brocq, Sabouraud, Darier, Milian, Lortat-Jacob and Louste carried on varied and multiple researches which advanced the renown of the Parisian school.

A clinical chair of dermatology and syphilology on the *Faculté de Médecine de Paris* was founded in 1880. Alfred Fournier was the first professor; he became universally known because of his remarkable work on syphilis. His book, "Syphilis and Marriage," was translated into many languages. That which immortalized the name of Alfred Fournier was his conviction that tabes and dementia paralytica are syphilitic in origin and that in the great majority of cases these diseases result from insufficient antisyphilitic treatment. Fournier was succeeded by Gaucher, Jeanselme and Gougerot.

It is expected that the war has only temporarily interrupted the brilliant course of French science.

LAYMON, Minneapolis.

LYMPHADENOMA: ITS ETIOLOGY AND ITS SKIN-LESIONS. E. C. WARNER, Brit. J. Dermat. **56**: 129 (May-June) 1944.

Lymphadenoma is essentially a disease of the reticuloendothelial system which usually manifests itself first in the lymph nodes, subsequently spreads to involve almost any organ in the body and, as a result of multiple lesions in many different organs, usually terminates in death from cachexia.

The author believes that etiologically lymphadenoma stands in an intermediate position between the granulomas and the generally accepted neoplasms.

The cutaneous lesions of this disease are of various types. Pruritus usually starts on the front of the legs or dorsa of the feet or, when generalized, is most severe in those areas. Herpes zoster is present in approximately 30 per cent of patients during some stage of their disease and usually is not associated with other signs of a lesion of the spinal cord. Papular lesions, especially on the trunk, appear in a small proportion of patients and may become vesicular. Ulceration of the cutaneous surface is seen, usually of the suprasternal and supraclavicular areas or of the upper part of the neck. In addition, generalized erythroderma is a fairly frequent manifestation of Hodgkin's disease.

THE RETICULAR TISSUE AND THE SKIN. A. H. T. ROBB-SMITH, Brit. J. Dermat. **56**:151 (July-Aug.) 1944.

In considering the eruptions associated with diseases of the reticular tissue, one should distinguish between those that are toxic manifestations and those that are resultants of specific cellular infiltrations. Of the former, purpura almost invariably is secondary to a hemorrhagic diathesis. Another nonspecific lesion is herpes zoster, which is peculiarly common, especially in patients with Hodgkin's disease. Pruritus without a specific lesion also is common in Hodgkin's disease, but in the patients with leukosis it is usually associated with a cellular infiltration and erythroderma.

The specific cutaneous lesions of the leukoses are most common in lymphoid leukosis, and may consist of (1) a diffuse erythroderma with a variable degree of scaling or (2) purplish nodular tumors affecting the face or (3) small nodules usually scattered over the body but sometimes limited to the conjunctiva or eyelids.

In myeloid leukosis specific cutaneous lesions appear much less frequently and usually develop as purplish nodular tumors that may be either limited to the face or widespread over the body.

Monocytic leukemia, the least common form, frequently presents specific cutaneous manifestations. The lesions may appear either as a diffuse eruption of bluish, slightly raised macules or as firm pale nodules deeper in the skin. The lesions may be transient and often may precede the development of the characteristic oral lesions.

In Hodgkin's disease, although symptomatic cutaneous manifestations (such as pruritus, nonspecific pigmentation, herpes zoster) are common, true involvement of the skin is rare. The lesions are usually on the trunk in the form of plaques which become ulcerated.

BLUEFARB, Chicago.

CONTACT DERMATITIS IN A MORPHINE FACTORY. S. ERNEST DORE and E. W. PROSSER, Brit. J. Dermat. **56**:177 (July-Aug.) 1944.

The authors report on nine patients with contact dermatitis, all of whom were workers in a morphine factory. Seven were men (out of a total of the 16 men employed) and 2 were women (all the women employed). The average period between commencement of employment and development of the first symptoms was fourteen and a half weeks.

One patient was engaged in the process of evaporation, crystallization and purification of "Gregory salt" (mixed morphine and codeine hydrochlorides); 3 in the process of separation of alkaloids and purification of separated morphine salt, and 5 in the process of drying, milling and packing of morphine salt.

In practically all of the patients the eruption began as an irritable erythematous dermatitis of the eyelids and surrounding area, sometimes accompanied with severe edema. Other regions often affected were the nape of the neck and the under surface of the chin in women, and the collar region in men. The arms and hands became involved later. In 1 man the scrotum became involved, and in another the dermatitis eventually became generalized. Otherwise the eruption remained limited to the exposed parts of the body (face, neck, arms and hands). The clinical appearance of the eruption varied considerably in different cases: On the

face and neck it was generally diffuse and of typically eczematous contact type, while on the hands it was sometimes like pompholyx. In 1 case it resembled erythema multiforme. Secondary infection occurred in several cases and lichenification of the skin from scratching in 1 case.

It has not been possible to determine the specific sensitizing agent. The involvement first of the eyelids with an edematous and erythematous type of reaction suggests that the irritant is air borne, as fine dust or spray. To support this is the fact that catarrh of the upper part of the respiratory tract occurred in most cases.

DIAGNOSIS AND TREATMENT OF LESIONS DUE TO VESICANTS. W. E. CHIESMAN, Brit. M. J. **2**:109 (July 22) 1944.

In a detailed report on the vesicant gases, the author makes the following recommendations:

Protection. Remain under cover and protect the face with a gas mask. Clothing contaminated by liquid should be removed at once. If the skin is also contaminated the liquid should be removed mechanically and the skin treated with antigas ointment or bleach cream and then washed. Washing with soap and water alone may spread contamination. Antigas ointments are all cutaneous irritants and must be washed off. The antidote for lewisite on the skin is hydrogen peroxide (20 volume).

Treatment of Skin. Do not use any prophylactic treatment when the skin is showing actual signs of damage, unless the presence of lewisite is suspected by early cutaneous changes. The value of BAL (a secret formula which is said to be remarkably effective in the treatment of arsenic poisoning) is considerable at this stage. Hair should be clipped from the affected areas and the part cleaned with a bland antiseptic. Try to keep blisters intact. Redundant skin should not be removed unless its removal is unavoidable. Apply a sterile oily dressing. Sulfonamide compounds can be used to counteract infection. Any form of coagulant treatment is contraindicated. Never change a dressing unless absolutely necessary: The exudate from lesions caused by mustard gas or by lewisite is innocuous.

SOME COMMON PAEDIATRIC PROCEDURES. DOYNE BELL, Brit. M. J. **2**:157 (July 29) 1944.

Among other procedures, the author discusses the treatment of strawberry nevi, maintaining that there is no exception to the rule that nevi which grow rapidly during the early months of life subsequently retrogress and, of their own accord, disappear on the average in about the fifth year.

SCURVY: A SURVEY OF FIFTY-THREE CASES. R. B. McMILLAN and J. C. INGLIS, Brit. M. J. **2**:[illegible] (Aug. 19) 1944.

In a general discussion of scurvy in adults found in an Edinburgh municipal hospital, the authors presented 53 cases of the "bachelor" type, for which they gave three principal causes: first, ignorance, mainly in men, of the need for potatoes and vegetables in the diet; second, apathy, leading to neglect of the same items because they required preparation and cooking; third, poverty, making it impossible to buy the food required for an adequate diet.

The authors feel that more than propaganda is needed for the elimination of scurvy, and it is suggested that local authorities take practical steps toward the provision of clean, cheap lodgings with canteen facilities under discreet supervision.

FATAL PURPURA AFTER SULPHAPYRIDINE. SHEILA SHERLOCK and J. C. WHITE, Brit. M. J. **2**:[illegible] (Sept. 23) 1944.

In reporting a fatal case of acute purpura occurring during a course of sulfapyridine therapy for pneumonia, the authors emphasize the vascular lesions as a basis for its cause. In view of the high mortality, all patients receiving sulfonamide drugs should be watched for extraneous bleeding.

SHAW, Chattanooga, Tenn.

SYPHILIS IN PREGNANT WOMEN. T. DIXON HUGHES, M. J. Australia **2**:273 (Sept. 9) 1944.

In a study based on the routine Wassermann and Kahn tests performed on 28,924 pregnant women, 160 had positive reactions, an incidence of 0.55 per cent.

HENSCHEL, Denver.

Correspondence

IMMUNIZATION THERAPY OF WARTS

To the Editor:—Dr. Hans Biberstein is responsible for a paper, "Immunization Therapy of Warts" (ARCH. DERMAT. & SYPH. **50**:12 [July] 1944). In his paper Dr. Biberstein quotes me on page 12 as saying: "Findlay's statement that warts of one species . . . are not referable to another species was refuted by Frank Schulz and others" (Schulz, F.: Experimentelle Uebertragung von Verrucae vulgares vom Rinde auf den Menschen mit ausserordentlich langer Incubation, Deutsche med. Wchnschr. **34**:423, 1908).

On page 15, Dr. Biberstein states: "Fifteen patients had condylomata acuminata; 9 of the 10 (90 per cent) who could be reexamined were 'uninfluenced', [sic] 7 who received up to fifteen injections were 'influenced.' This refutes the second part of Findlay's statement that extracts of warts have no curative effect in animals of a different species."

Dr. Biberstein has obviously not done me the honor of reading my original paper, since the only reference to Findlay is "cited by Rulison" (Warts: A Statistical Study of Nine Hundred and Twenty-One Cases, ARCH. DERMAT. & SYPH. **46**:66 [July] 1942).

This omission on Dr. Biberstein's part is the more curious in view of the fact that the true reference to my paper is Findlay, G. M.: Warts, in Andrews, C. H.; Arkwright, J. A., and others: A System of Bacteriology in Relation to Medicine, London, His Majesty's Stationery Office, 1930, vol. 7, p. 252. The "System of Bacteriology" is, to my knowledge, available in three medical libraries in New York and is a well known standard work.

If Dr. Biberstein will take the trouble to refer to my article, he will find no such categoric statements as he attributes to me: On page 257 there is a paragraph, "The Relation of Human and Animal Warts," in which Ullman's results (1923) in producing a papilloma in the vagina of a dog by inoculation with material from a laryngeal papilloma are mentioned, as well as the failure of Serra (1924) to infect laboratory animals with human warts. An account of my own experiments states that "dog warts failed to infect man, rabbits, mice or guinea pigs, as did bovine warts. Human warts were not infective for dogs, rabbits, rats, mice or guinea pigs." At the time my paper was written this was a fair summary of the position, since the experiments of Schulz (1908) are entirely uncritical.

For the second statement which Dr. Biberstein attributes to me there is no evidence whatsoever. I have never used extracts of warts in attempting to cure warts in animals of any species and know nothing of their effects.

The paragraph headed "Immunity" in my article on warts, Dr. Biberstein may find of interest.

For the sake of scientific accuracy, I should be much obliged to Dr. Biberstein if he would draw the attention of your readers to the errors in his paper.

G. M. FINDLAY, Brigadier,
A.M.S., West African Force.

EPIDERMOLYSIS BULLOSA

To the Editor:—In their article (ARCH. DERMAT. & SYPH. **47**:647 [May] 1943) Franks and Davis described 4 cases of epidermolysis bullosa occurring in soldiers and made the statement that "No case of this type ['. . . a rare hereditary type with the bullae limited to the feet'] has previously appeared in the American literature." This statement of Franks and Davis was in error in that we had previously described such a case in our article "Epidermolysis Bullosa Hereditaria: Report of Two Cases with Extensive Family Histories" (ARCH. DERMAT. & SYPH. **46**:419 [Sept.] 1942).

Samitz (ARCH. DERMAT. & SYPH. **48**:159 [Aug.] 1943), Waisman (*J. A. M. A.* **124**:1247 [April 29] 1944), Greenberg (ARCH. DERMAT. & SYPH. **49**:333 [May] 1944) and Mooney (ARCH. DERMAT. & SYPH. **50**:167 [Sept.] 1944) apparently had no difficulty in finding and citing our cases, although several other authors omitted citation of our publication, perhaps because they followed the incomplete references of Franks and Davis.

We have thought it worth while to call these omissions to your attention, as this form of epidermolysis is not only of dermatologic and genetic interest but of particular military importance; as shown in our article, Sulzberger and we first called attention to the military significance of this form of eruption of the feet.

MORRIS LEIDER, Lieutenant Commander (MC), U.S.N.R.
RUDOLF L. BAER, M.D., New York.

Society Transactions

NEW ENGLAND DERMATOLOGICAL SOCIETY

JACOB H. SWARTZ, M.D., *President*

FRANCIS M. THURMON, M.D., *Secretary*

Boston, Feb. 9, 1944.

A Case for Diagnosis (Acrodermatitis Continua [Hallopeau]?). Presented by DR. JOHN ADAMS, Boston.

M. A., a white woman, a 41 year old secretary, complains of an eruption intermittently affecting the entire body (exclusive of the face) for eight years. At the onset, the skin at the base of the neck anteriorly became red, and the area enlarged rapidly to cover practically the entire cutaneous surface. Pustular lesions appeared, and painful crusts followed. This eruption was accompanied with a low grade fever, malaise and loss of weight. The first attack lasted three months. Subsequent outbreaks occurred three to six times yearly; each attack lasted two weeks to three months. For the past eight months areas have persisted on her right forearm, wrist and forefinger. Previously, the skin had been normal between these episodes, and the patient had experienced good health.

Examination reveals sharply circumscribed plaques of deeply reddened and thickened skin with scattered pustules and large flakes of dry, purulent exudate. These are seen on the extensor surfaces of the arms, on the trunk, particularly along the right lower portion of the thorax, over the hips and on the right leg. The right forefinger is completely involved. On the scalp there are erythematous plaques 1 to 3 cm. in diameter covered with a white scale. The toe nails are yellow and hypertrophied. The hands, feet and face are clear.

The urine was normal. Hematologic studies showed: hemoglobin content 12 Gm.; erythrocytes 5,320,000; leukocytes 12,100 and a differential count of 71 per cent polymorphonuclears, 19 per cent lymphocytes 4 per cent monocytes and 4 per cent eosinophils. Cultures of materials from the crusted areas yielded Staphylococcus aureus.

The oral administration of sulfathiazole caused nausea, vomiting and a febrile reaction. Additional therapy has comprised saline baths and use of an ointment of petrolatum and hydrous wool fat.

DISCUSSION

DR. BERNARD APPEL, Lynn, Mass.: I could not help observing how closely the lesions on the right elbow resemble psoriasis. There is a moderate amount of typical white, silvery light fluffy scales. I did not scratch deep enough to get pinpoint bleeding. This fairly typical area of psoriasis extends directly and almost imperceptibly into that portion which shows the greatest amount of acute inflammation with the closely placed pinpoint pustules. The eruption strongly suggests a diagnosis of pustular psoriasis.

DR. FRANCIS M. THURMON, Boston: The eruption at first glance does not resemble psoriasis. I have never seen psoriasis with atrophy of the subcutaneous tissues, such as is present on the leg of this patient. Early atrophic changes have begun to involve the forearm and wrist.

DR. JOHN G. DOWNING, Boston: The differentiation between these two diseases, acrodermatitis continua and pustular psoriasis, is difficult. In both entities pustular lesions occur and are particularly well defined on the chest. A distinguishing factor is the involvement of the extremities. "Acro" is the root word meaning "extremity." In acrodermatitis the primary sites of the involvement are the terminal phalanges. I prefer the diagnosis of acrodermatitis in this case to that of pustular psoriasis. The eruption does not have the shiny dry scaly appearance characteristic of psoriasis. I shall be interested to see what happens with administration of sulfapyridine.

DR. FRANCESCO RONCHESE, Providence, R. I.: Have any patients with pustular psoriasis been treated with sulfapyridine?

MAJOR EARL A. GLICKLICH, M. C., A. U. S.: How long can one continue to give sulfonamide compounds? Apparently this woman has received this therapy for over six months.

DR. WALTER F. LEVER, Boston: Dr. Swartz and I started treating patients with dermatitis herpetiformis with sulfapyridine in 1939. Two of these patients have been taking from 2 to 3 Gm. of sulfapyridine daily for more than four years. Studies of the blood have been carried out at regular intervals, and no harmful effects have been noted.

DR. G. MARSHALL CRAWFORD, Brookline, Mass.: This woman has lesions on her trunk as large as the hands, which resemble those on the arms and legs. The skin on her feet, toes and knees shows changes compatible with psoriasis. The toes are not affected in the same way as the fingers. Considering only the involvement of the toe nails, one might be justified in considering this psoriasis. However, the picture as a whole strongly suggests acrodermatitis.

DR. FRANCIS M. THURMON, Boston: Dr. McCarthy called attention to the tongue. I wonder if he has anything further to add.

DR. FRANCIS P. MCCARTHY: I observed that this patient's tongue was practically denuded. There was an intensification of the remaining markings. One or two islands of normal papillae lay near the tip, and the color was within normal limits. The picture resembled a glossitis totalis. If the dermatologist would look into the oral cavity as a part of the routine examination, he would note many interesting lesions.

Is the blood level of the sulfonamide compound sufficiently high with these small doses of the drug used over a long period of time to be of any real value? There may be established a tolerance to the sulfonamide drug. In such patients an acute hemolytic anemia may rapidly develop. There is an element of danger in the treatment, and a great responsibility is assumed by the dermatologist who continues sulfonamide therapy indefinitely. The patient might consider taking extra tablets independent of the prescribed dose.

DR. C. GUY LANE, Boston: The only comment I wish to make in respect to Dr. McCarthy's remarks is that I fully appreciate the risks and dangers of sensitization and of perhaps serious results associated with sulfonamide therapy. Because of the pruritus, persons with dermatitis herpetiformis lose their appetites, are below par physically and cannot sleep. Since they are relieved by relatively small doses of the sulfonamide drug, I am inclined to go ahead with this therapy and assume whatever risk may be involved. The 2 patients who have been taking the drug for

two years are comfortable, are not troubled by itching and do sleep. I have not been able to find a substitute that will give them the comfort they demand.

A Case for **Diagnosis (Dermatitis Factitia; Dermatitis Medicamentosa?).** Presented by DR. JOHN ADAMS, Boston.

M. S., a white woman, a 27 year old defense worker, complains of an eruption on the thighs and legs of two years' duration. The dermatitis is characterized by exacerbations and remissions at approximate intervals of two weeks. Eight months ago, for several weeks, similar lesions appeared on the neck and arms. On alternate days for the past five years she has chewed Feen-a-mint. Four months ago after a hemorrhoidectomy she avoided Feen-a-mint, and during this period there was a decided improvement in the eruption.

The eruption is characterized by multihued lesions resembling healing ecchymoses. The lesions are of irregular size and shape, varying from 2 to 10 cm. in diameter. All areas are slightly raised and tender to pressure. Some are firm; others, soft.

A patch test with phenolphthalein produced no reaction.

DISCUSSION

DR. FRANCESCO RONCHESE, Providence, R. I.: Why is a diagnosis of dermatitis factitia considered in this case?

DR. C. GUY LANE, Boston: The diagnosis was questioned when, during the rounds of the ward recently, the patient gave a history of repeated trauma to one knee. Persistent lesions developed at the site of this trauma. Later additional lesions appeared on the thighs and legs but not on the upper part of the body. Dr. Adams raised the question of trauma from pounding. We had no reason to suspect that these lesions were self inflicted, but they did appear soon after and at the sites of the trauma. Although there were miliary lesions which were relatively difficult to explain on the basis of a medicamentosa, it is difficult, on the other hand, to account for these lesions entirely on the basis of trauma. For a long time she had habitually taken Feen-a-mint and when not taking Feen-a-mint had noticed a remission of the lesions. She was given 2 grains (0.13 Gm.) of phenolphthalein, and the reaction was much less severe than I should have expected. The eruption certainly is not as quiescent as it was a week ago. Today the lesions are rounded, slightly elevated, and definitely more palpable and erythematous. I am not willing to subscribe to the diagnosis of dermatitis factitia, but the whole matter is certainly not explained by the phenolphthalein.

DR. GEORGE E. MORRIS, Boston: May I suggest that capillary fragility could be aggravated by phenolphthalein. The lesions resemble a phenolphthalein eruption. A moccasin venom test should be made before and after the administration of phenolphthalein to see if the reaction is intensified by the medication.

DR. ALFRED HOLLANDER, Springfield, Mass.: I believe that one should call this eruption dermatitis medicamentosa. On palpation the leg presents a band-like swelling which suggests urticaria. A year ago I saw a man who presented bullous hemorrhagic lesions on the dorsa and palms of the hands. These bullous lesions suddenly disappeared, and four or five months later typical urticaria developed, which could not be controlled. Last week I learned that after discontinuing all medication he was completely relieved. Perhaps an allergy or a sensitivity to certain drugs should be considered in such cases.

DR. JOSEPH GOODMAN, Boston: It seems to me there are certain aspects of this case which strongly suggest an atypical form of erythema nodosum, such as the color changes, the persistent eruption, the tenderness and the limitation to the legs. A biopsy might be enlightening.

DR. WILLIAM P. BOARDMAN, Boston: For my own information, I should like to know if this patient was given a patch test with phenolphthalein.

DR. JACOB H. SWARTZ, Boston: I am aware of the report of Dr. Wise and Dr. Sulzberger on patch tests for fixed eruptions (ARCH. DERMAT. & SYPH. **27**:549 [April] 1933), but I thought that such a test might be worth trying in this case, since the eruption is not purely fixed but is a mixed eruption.

DR. JOHN G. DOWNING, Boston: Dr. Sulzberger stated that a patch test with phenolphthalein is useless in diagnosing a dermatitis due to phenolphthalein. He does mention the fact that it is only in the eczematoid eruptions of dermatitis medicamentosa that the patch test is of value.

DR. ALFRED HOLLANDER, Springfield, Mass.: I have never seen a phenolphthalein test produce swelling which penetrates the deeper tissue. Often this type of dermatitis medicamentosa is a fixed eruption.

DR. FRANCIS M. THURMON, Boston: In palpating these areas which apparently were ecchymotic, I noticed thin lines, which were slightly beaded as though there might possibly be a low grade inflammatory reaction in the veins or lymphatics. I suggest that the biopsy be deep enough to include the underlying vessels. I suspect that these lesions are connected with the vascular supply.

Necrobiosis Lipoidica Diabeticorum. Presented by DR. LEON BABALIAN, Portland, Maine.

This case was presented by means of a case history and colored lantern slides.

D. N., a white American salesman aged 29, complained of a lesion on the front of the right leg of four years' duration.

He had been known to have diabetes mellitus since 1934. Since that time he has had continuous insulin therapy but has not always adhered closely to his prescribed diet. The cutaneous lesion appeared first in 1936 and has been slowly progressing.

Examination reveals a well defined oval plaque 5 by 11 cm., of a pinkish yellow color with erythematous border. The plaque is generally depressed except for its margins, which are slightly elevated. Telangiectases are present. In the center of the plaque is an irregular necrotic ulceration 2 by 4 cm. The entire lesion is firm and painless.

The urine was normal. The blood sugar was 110 mg. and the blood cholesterol 166 mg. per hundred cubic centimeters.

DISCUSSION

DR. C. GUY LANE, Boston: I should like to say a word here about the lifelike qualities of that Kodachrome still projection. I was almost afraid to touch the picture lest I might rub off some of the serum on my finger. It offers a prospect for teaching and demonstrating cases which would be valuable in the absence of the patient. I wish to congratulate Dr. Babalian.

DR. LEON BABALIAN, Portland, Maine: Necrobiosis lipoidica diabeticorum has been described since the advent of insulin. The general belief is that this entity is related to uncontrolled diabetes. The case presented today is especially interesting because the condition appears in a person with diabetes who has been treated with continuous insulin therapy. In spite of this therapy, the disease has progressed.

On the other hand, it is interesting to note that hepatic and arterial manifestations of fat dysfunction are found with greater frequency in cases of diabetes since the advent of insulin therapy (Dragstedt, L. R.: The Present Status of Lipocaic, *J. A. M. A.* **114**:29 [Jan. 6] 1940). I think that this also has a dermatologic application in that necrobiosis lipoidica diabeticorum should be treated not with insulin but with lipocaic.

DR. C. GUY LANE, Boston: One point occurred to me which I neglected to mention, namely, that the ulceration has been present for three months. There is an open ulceration which I estimate to be 3 cm. in diameter, perhaps even slightly larger. The inner border appears somewhat infiltrated. It is known that scar or atrophic tissue is apt to be present, and I wonder whether in this instance an excision and skin graft should be done, especially if that ulceration persists. I do not know of nor have I seen anything in the literature with regard to excision of necrobiotic lesions. If the lesion persists or becomes enlarged, the possibility of cancer should be considered.

DR. JACOB H. SWARTZ, Boston: From what part of the lesion was the biopsy specimen taken? Was it taken before the ulceration appeared?

DR. LEON BABALIAN, Portland, Maine: The biopsy was made when the ulceration was present, and the specimen was taken from the border of the plaque.

Herpes Gestationis. Presented by DR. JACOB H. SWARTZ, Boston.

E. B., a Polish-American housewife aged 37, presents an eruption of eight weeks' duration.

In early December 1943, during the seventh month of her fifth pregnancy, erythematous papules accompanied with intense itching appeared on the midflexor portion of her thighs. During the following weeks papules appeared and coalesced on the arms, calves and trunk. By late December vesicles appeared on the thighs and rapidly increased in size to form bullae 1 to 3 cm. in diameter. The intense itching could not be controlled. By early January a few of the bullae became hemorrhagic, and additional bullae appeared over the ulnar portions of the arms. In late January the patient delivered a live healthy boy, and after the delivery the eruption completely disappeared.

Examination during the eighth month of pregnancy revealed patches of thickened erythematous, lichenified and excoriated skin on the flexor surfaces of the thighs and forearms. Papules were scattered over the calves, arms and trunk. Large bullae, 1 to 3 cm. in diameter, were clear or hemorrhagic; heavily crusted lesions were present on the thighs and forearms.

The laboratory findings prior to delivery were irrelevant.

Treatment comprised denuding the bullae and applying a 2 per cent solution of basic fuchsin to the denuded areas. A calamine lotion also was applied. Over a ten day period she was given sulfapyridine, 3 Gm. daily.

DISCUSSION

DR. JACOB SWARTZ, Boston: This patient did not respond to sulfapyridine as favorably as other patients with dermatitis herpetiformis who have been treated with this drug.

DR. WALTER F. LEVER, Boston: A few years ago, a patient with a generalized bullous eruption was seen at the Massachusetts General Hospital. The eruption had begun in the fifth month of her pregnancy and persisted until shortly after delivery. It was indistinguishable from pemphigus. Pemphigus-like eruptions occur in pregnancy. They have been described sometimes under the name of pemphigus and sometimes under the name of herpes gestationis.

DR. LEON BABALIAN, Portland, Maine: I think the difference between pemphigus and herpes gestationis is that in the latter the vesicles or bullae are arranged in clusters. The case presented today is described as showing clustering of the lesions, such as is present in herpes gestationis. That is quite different from pemphigus.

DR. JOHN G. DOWNING, Boston: At St. Elizabeth's Hospital, Brighton, I frequently see patients who have been treated for scabies. They present a generalized vesicular eruption and at times show bullae. At the present time I am attending a multipara six months pregnant, who for the past two months has shown vesicles and bullae which completely cleared within ten days. A generalized urticaria then developed. After the involution of this eruption, there developed on the flexor surface of one arm a large patch, 5 by 20 cm., which was elevated and flat and composed of pea-sized vesicles. I gave her a simple local application and requested that she report the progress of these lesions. She reported that they disappeared. They had dried and desquamated. In this syndrome, one may see all types of eruption, varying from urticaria to the bullous type of erythema multiforme.

DR. JACOB H. SWARTZ, Boston: When I saw the patient soon after she delivered an 8 pound (3,630 Gm.) infant, the eruption had disappeared except for pigmented blotches marking the sites of previous lesions. The clinical picture coincided with Dr. Downing's description. The lesion in this case was of the bullous type which continued to develop up to the time of delivery.

DR. JOHN G. DOWNING, Boston: I saw a woman at the Boston City Hospital who had a severe bullous eruption of herpes gestationis. She was advised never to become pregnant again. However, she has had many more babies. In neither of these two pregnancies did she have any disturbance of the skin. Herpes gestationis does not necessarily recur during subsequent pregnancies.

DR. JACOB H. SWARTZ, Boston: This woman is a multipara, and this is the first time she has had herpes gestationis.

A Case for Diagnosis (Acquired Localized Trichorrhexis; [Trichoclasis]; Keratosis Pilaris?). Presented by DR. FRANCESCO RUSSO-CHESE, Providence, R. I.

A. N., a white American girl aged 16, presented a disturbance of the scalp of three years' duration.

She first noticed on the vertex of the scalp several round areas of thinning which were not clearcut spots of alopecia areata. The hairs in these areas became stiff and coarse and broke off approximately 1 inch (2.5 cm.) from the scalp. The hairs of the affected areas have remained in this condition, while the surrounding hairs of the parietal, occipital and frontal portions are of a normal wavy silky texture. She says that she has neither pulled nor cut the hairs.

Examination reveals three distinct types of hair. First, the hair along the frontal, temporal and occipital regions appears normal. Second, the hair in the affected areas is sparse and coarse. A moderate degree of fragility and no twists, constrictions, nodes or other alterations are found in the shaft. The roots are definitely abnormal; they are pointed and twisted. No fungi were found on direct examination of the hairs. Third, the hair between the involved hair and the normal hair is coarse, long and kinky. The hairs may be pulled in

alopecic areas without causing pain. The distal ends of the hairs are smooth, as though cut with shears. There is pronounced keratosis pilaris in the area of alopecia on the scalp and on the arms and legs. The skin of the calves and ankles shows a mild degree of ichthyosis.

value of 94 micrograms of carotene and 88 U. S. P. units of vitamin A per hundred cubic centimeters of serum. The nonprotein nitrogen was 36 mg., the cholesterol 170 mg. and the sugar, 93 mg. per hundred cubic centimeters of blood. The hemoglobin content was 71 per cent, erythrocytes numbered 5,100,000 and leukocytes

Acquired localized trichorrhexis (trichoclasis). (*A*) Apparently normal hair on the frontal hair line and back of the head. Low power photomicrograph of an apparently normal section from the back of the scalp. (*B*) Area of alopecia, keratosis pilaris and spontaneous breaking of the hair at short distance from the scalp. Thin, fragile hair with pointed and twisted roots. Low power photomicrograph from the alopecic area showing a thin and wavy epidermis, hyperkeratosis, indentations and absence of atrophic coil and sebaceous glands.

Cultures planted on Sabouraud's medium failed to yield fungi. The Hinton and Kahn reactions of the blood were negative. The blood showed a fasting

5,400, with a differential count of 68 per cent polymorphonuclears and 32 per cent lymphocytes. The urine was normal.

A specimen for biopsy was removed from the vertex of the scalp and from the occiput. The skin of the occiput showed normal epithelium and normal coil and sebaceous glands. The skin from the vertex showed a thin and wavy epidermis with hyperkeratosis and indentations, as seen in keratosis pilaris: the sebaceous and coil glands were absent or very small. This section in toto was fragmented and showed more friability under the cutting knife than is normal. The ensemble is consistent with a form of atrophy of the skin and its appendages.

NOTE.—On June 9, 1945, after fourteen months of intensive vitamin A therapy, although the keratosis pilaris has improved, the area of alopecia of the scalp has not changed.

DISCUSSION

DR. LEON BABALIAN, Portland, Maine: I could not see nodules on the hairs. Are you sure that it is not a question of simple trichotillomania? It would be difficult to extract the hair forcibly in large quantities. Usually trichorrhexis is limited to the parietal areas.

DR. J. HARPER BLAISDELL, Boston: I suggest the diagnosis of trichotillomania. In the cases that I have observed it occurred in adults who did not deny this vicious habit. Most persons with trichotillomania say there is an irresistible desire to pluck the hairs; this admission is an indication of their desire to be helpful.

DR. JOSEPH GOODMAN, Boston: I think that a feature of this girl's history which is of interest is the fact that the lesions she presented on the arms and legs appeared at the same time that her hair started to fall. I note that vitamin A in large amounts is to be administered. I suspect that it may be helpful for the cutaneous lesions and for the hair. I have been impressed by the fact that in certain cases of vitamin A deficiency, there may be a loss of hair.

DR. GEORGE E. MORRIS, Boston: The association of keratosis pilaris and monilethrix is well known. The eruption on the girl's arms is characteristic of keratosis pilaris. Therefore, hairs should be carefully and repeatedly examined for beading. It is well known that such beading may be localized to one or several spots of the scalp and also that remissions and exacerbations of the abnormality may occur.

DR. FRANCESCO RONCHESE, Providence, R. I.: The few hairs from the alopecic area that I had the opportunity to examine showed abnormal roots and fragile shafts. There were no nodes as in trichorrhexis nodosa. The hairs of the back of the head were apparently normal. This and the fact that the alopecic area is not on the temples or in irregular spots but occupies a uniform square area on the top of the head in a male baldness pattern are against trichotillomania.

A Case for Diagnosis (Premycosis Fungoides?). Presented by MAJOR EARL A. GLICKLICH, M.C., U. S. A.

B. F., a white man aged 30, complained of an itching eruption of four years involving the groins.

The eruption first appeared on both legs. Six months later both groins became involved. Pruritus of the involved areas is a constant accompanying symptom. The patient entered the military service in 1942 and since that time has experienced recurrent episodes of the eruption, resulting in several periods of hospitalization.

Examination reveals multiform lesions of varying sizes which are slightly indurated and erythematous and have slightly raised borders. With involution, pigmentation marks the sites of previous lesions.

Examination of the blood showed a hemoglobin content of 85 to 90 per cent, 4,500,000 erythrocytes and 5,000 to 8,000 leukocytes. The differential count was essentially normal except for an increase of eosinophils to 6 per cent. The sedimentation rate was normal. Direct smear and culture did not show fungi. A biopsy specimen was taken from the right groin. Histologic examination showed that the epithelium was thickened with moderate intracellular edema, slight hyperkeratosis, moderate elongation and clubbing of the papillae. The basal cell layer was sharply defined. There were moderate edema of the subepithelial tissue and predominantly a perivascular infiltration of the superficial layers of the cutis by eosinophils, lymphocytes, histiocytes and a few neutrophils.

DISCUSSION

DR. C. GUY LANE, Boston: I am interested in the suggestion about the primary stage of mycosis fungoides, and it seems to me that it is suggested by the persistent itching and some slight infiltration of the lesions in his left groin. The diagnosis can be made only by biopsy at a later date.

It would be interesting to see whether one could use roentgen irradiation as a diagnostic feature in this type of case. One could treat one or two lesions and see what change takes place after administration of 30 or 50 or 75 r. In my experience, a lesion of mycosis fungoides is apt to resolve much more rapidly than any other kind that I know of.

A Case for Diagnosis (Seborrheic Dermatitis?). Presented by DR. R. H. GOLDFARB, Boston.

B. L., a white Canadian housewife aged 52, presented an eruption involving the nose, lips and premalar areas of six years' duration. A papule first developed on the nasal septum, and it recurred intermittently until three years ago. At that time there appeared a swelling and erythematous infiltration of the nose and upper lip accompanied with scaling of the eyelids. This infiltration and edema gradually extended to involve the left cheek and ear.

Examination reveals a suffused erythema which circumscribed the nose, lips, adjacent parts of the cheeks and chin. There is no pitting edema. There is slight scaling of the left ear and adjacent portion of the scalp.

Laboratory examinations showed nothing significant.

Treatment has comprised use of moist boric acid compresses, boric acid ointment and aqueous solution of zephiran chloride locally. Potassium iodide and diethylstilbestrol have been administered orally without apparent benefit.

DISCUSSION

DR. GEORGE E. MORRIS, Boston: May I suggest the diagnosis of recurrent erysipelatous infection?

DR. JACOB H. SWARTZ, Boston: May I suggest the diagnosis of chronic lymphangitis. Removal of a focus of infection may result in the clearing of the eruption.

DR. GEORGE SCHWARTZ, Philadelphia: I saw this woman's eruption three or four years ago during the acute stage. She had a temperature each afternoon of 99.2 and 99.4 F. I administered sulfathiazole. While she was taking this drug, she had no recurring attacks during the time I saw her.

Dermatitis Venenata of the Axillas. Presented by DR. FRANCIS M. THURMON, Boston.

B. B., a white girl aged 17, employed in a dental clinic, presented lesions of five months' duration in each

xilla. She had used a proprietary deodorant, and afterward a papular eruption developed which burned. Examination reveals papular lesions of follicular distribution confined exclusively to the hairy portions of each axilla. The cutaneous markings are exaggerated and present a slightly thickened appearance. There is no evidence of infection. The axillary lymph nodes are not enlarged.

Treatment comprised use of moist boric acid dressings, antipruritic lotion and boric acid ointment.

DISCUSSION

DR. BERNARD APPEL, Lynn, Mass.: It seems to me that this is Fox-Fordyce disease in an early stage. I questioned the patient about lesions on the nipples and the pubis; she denied their presence. I did not examine these areas. The eruption is limited to the axillas. In my opinion, the uniformity of the papules and their relation to the hair follicles is characteristic of this disease. Apropos of this particular discussion, I should like to recall the case which I presented at a previous meeting. The patient was a girl with Fox-Fordyce disease whom I had treated with roentgen therapy, to which a resistance eventually developed, with intractable pruritus. The family physician referred her to Dr. Lane for consultation; he agreed with my observations. However, the family physician referred her to Dr. Eugene Traub of New York, who advised dissection of the axillas. This treatment seemed radical. I should be careful of the prognosis in the case presented here today.

DR. FRANCIS M. THURMON, Boston: When I first saw this patient, I immediately thought of Fox-Fordyce disease. If it is this disease, it is in the earliest stage I have seen. That diagnosis I felt was at least questionable, since the lesions, which were confined to the axillas, almost disappeared with the use of soothing topical applications. Then, in order to preserve the lesions for today's presentation, a slightly irritating ointment was applied.

DR. JOHN G. DOWNING, Boston: My diagnosis is Fox-Fordyce Disease.

DR. ALFRED HOLLANDER, Springfield, Mass.: Should not one consider lichen simplex chronicus (Vidal), which is characterized by remissions and exacerbations? With the application of a tar ointment it becomes acutely inflamed.

Favus (Involving the Scalp, Nails and Glabrous Skin in Four Generations). Presented by DR. J. H. SWARTZ and DR. ETHEL M. ROCKWOOD, Boston.

P. C., aged 6, a white American schoolboy, presents an eruption of the scalp which has been present since he was 18 months old and which has been treated without success.

His great grandfather had a disturbance of the scalp following an injury in childhood. The grandmother since the age of 10 years has had lesions of the scalp and finger nails. The mother had lesions of the scalp. Other relatives have not been examined.

Examination shows numerous patches throughout the scalp characterized by loss of hair, yellowish crusting and atrophy. There are numerous yellowish crusts, some of which are surrounded by erythematous areolas on his face. A characteristic "mousy" odor emanates from the patient. On the mother's scalp there is a silver-dollar-sized area consisting of central atrophic scarring and peripheral yellowish crusting. The grandmother shows no active lesions of the scalp but all of her finger nail beds are involved, being deformed and of yellowish white discoloration.

Microscopic study of the material obtained from the involved hairs of the patient and of his mother, together with the finger nails of the grandmother, showed chiefly mycelium and also the reproductive stage of the fungus. Organisms cultured from the facial lesions showed grossly the characteristics of Trichophyton schoenleini. The Wood filter test of the lesions of the scalp showed golden yellow fluorescence.

None of these patients have been treated.

DISCUSSION

DR. JACOB H. SWARTZ, Boston: The lesion of the great grandfather's scalp developed when he was a young man, after an injury, according to his daughter. The grandmother's lesions of the scalp and nails developed afterward. The mother's lesions of the scalp appeared when she was a youngster. This child has had his lesions since the age of 2, and recently lesions on the face developed. The grandmother has had the lesions of the nails since shortly after the lesions of the scalp developed, when she was 10.

DR. ADRIAN H. SCOLTEN, Portland, Maine: I should like to ask Dr. Swartz a question. When one handles these patients with favus, is one in any danger of contracting favus?

DR. JACOB H. SWARTZ, Boston: I have handled quite a number of them, and I frequently get enthusiastic and am not too careful. So far I have not contracted favus. There is a certain predisposition to this infection; otherwise one cannot explain its presence. In this case the grandmother had sisters, and, as far as she knows none of them have had the infection. It would be interesting to see if the child's brothers or sisters contract favus. It is contagious—how and why, I do not know.

DR. JOHN G. DOWNING, Boston: My most interesting experience with favus was in Morocco. I was passing through the native quarter. There were two little boys fighting in the street. One knocked the fez off the other's head, and I saw scarring with alopecia of the scalp. I gave the youngster 10 cents to pose for a picture. Soon we heard what sounded like a riot behind us. There were about 50 children running down the street, and they all wanted their pictures taken. They took their fezzes off, and all who wore fezzes had ringworm. I was able to get eight Kodachrome pictures showing the various stages of favus.

DR. JACOB H. SWARTZ, Boston: What I meant was that the ordinary handling of a patient with favus does not involve high risk of contagion. In certain countries and with certain populations, the disease is not highly contagious. In this respect, favus is similar to leprosy. I recall a girl in Chelsea, Mass., who had favus of the entire scalp. She had brothers and sisters, and no member of the family had it except the grandmother, who had favus of the nails.

DR. ADRIAN H. SCOLTEN, Portland, Maine: Is it the opinion that people in this country are more likely to contract favus than people in other countries?

DR. ALFRED HOLLANDER, Springfield, Mass.: Why do American dermatologists see so few cases of favus as compared to physicians in the European countries? In Europe it is highly endemic, especially in Russia. After 1920, favus in children was endemic in the Rhineland. On the whole, the average American worker is cleaner than the average European worker, and this cleanliness may be a factor explaining fewer cases here.

DR. FRANCESCO RONCHESE, Providence, R. I.: Those epidemics in Europe are among children. Favus in adults is rare.

DR. ADRIAN H. SCOLTEN, Portland, Maine: May I ask what Dr. Swartz is going to do with these children?

DR. JACOB H. SWARTZ, Boston: I have not decided. In my experience the results of epilation have been disappointing, since the infection recurs and the epilation has to be repeated. I suggest a combination of roentgen epilation and ethyl iodide inhalation.

DR. EDWARD A. LAFRENIERE, Arlington, Mass.: What roentgen dosage do you use for epilation?

DR. C. GUY LANE, Boston: We used 280 r with 100 kilovolts and 10 milliamperes. We were using 300 r in a series of cases in which there was 100 per cent epilation. When one produces 100 per cent epilation, one never knows if the favus is entirely destroyed or not. With 270 r we did not have quite enough epilation. I have been accustomed to consider 300 r as an erythema dose. To me, 300 r, epilation dose and 1 skin unit are synonymous. That definition is not absolutely true, for the dose will vary slightly with each patient, but I feel that the epilation dose of 280 r is the safest measure.

DR. WALTER T. GARFIELD, Boston: How many times may one safely epilate such patients with recurrent attacks?

DR. C. GUY LANE: I think that I have treated 1 patient in this way three times. The epilation was performed after a period of six months intervened. I do not like to do that.

A Case for Diagnosis (Pustular Bacterid; Pustular Psoriasis?). Presented by DR. W. T. GARFIELD, Boston.

G. F. D., a white American man, aged 30, a clerk, has for the past nine years had a scaly eruption on his right palm. The entire palm showed a slight infiltration, white flaky scales and an erythematous base.

DISCUSSION

DR. J. HARPER BLAISDELL, Boston: I am asking for the discussion of this case for the benefit of Dr. Downing as well as for my own benefit. In the first place, I should like to raise the question, has the man psoriasis? Is it possible to have a single palmar lesion of psoriasis of many years' duration? The man says that he does not have dermatitis elsewhere. In the second place, if it is not psoriasis, what is it? In the third place, is it aggravated by his occupation as a freight handler? His hands are subject to trauma.

DR. JOHN G. DOWNING, Boston: It is a question of accepting certain definite clinical entities or refusing to accept these entities. They have been described in the literature and are fairly definite in their character. As has been noted, that man had the eruption on one palm. It was sharply outlined and reflected the light; it was shiny in character and had a shiny scale and the typical brilliant red base. I first saw him as a private patient and advised him not to spend any more money, because the lesion was refractory to treatment. At that time there were typical white vesicles, which dried and became yellowish and left a dark crust or heavy scale. To my mind, that is the picture of the type of eruption which has been described by some authors as pustular psoriasis. It has also been described by George C. Andrews as a recalcitrant pustular bacterid occurring on one palm or on one sole. It may be accompanied with scaly patches or with other types of eruption, and in such cases I prefer the diagnosis of pustular psoriasis. In cases in which there is no accompanying patch of psoriasis, I prefer Andrews' description.

I have had, in my own practice, cases of this type in which the patients were laborers, teachers and clerks. There is the industrial aspect—whether or not the man's work as a freight handler could be the precipitating factor of this condition. It is known that a freight handler suffers considerable trauma to the palm. Whether he were a freight handler or a school teacher or a clerk, he might have this eruption. The most persistent eruption of the sort that I have seen was in a hotel manager. He did no manual labor. Two weeks after his tonsils were removed, the eruption disappeared.

We will try sulfapyridine for this man.

DR. JACOB H. SWARTZ, Boston: I should like to suggest the diagnosis of infection with Trichophyton rubrum. Here is a unilateral lesion, fairly well defined, of nine years' duration. I should like to know whether cultures for Trichophyton rubrum have been made, since this organism could produce this very picture.

A Case for Diagnosis (Sarcoid; Tuberculosis Miliaris Disseminata Facei?). Presented by DR. G. MARSHALL CRAWFORD, Boston.

B. S., a white American woman aged 77, complains of lesions on the face of eight months' duration. One year ago she had iritis of the right eye. Eleven months ago an eruption of the erythema multiforme type appeared on the dorsa of the hands. Ten months ago herpes zoster involved the left scapular region and adjacent portion of the arm. Eight months ago a red macule appeared near the right ala nasi. Shortly thereafter a row of tiny pearly nodules involved the chin. The latter grew extremely slowly, and additional nodules appeared from time to time on various parts of the face. When seen four months ago, these nodules resembled shiny pink pearls. Those on the chin were in an absolutely straight transverse line. Some had a tiny scale on top, while others were dimpled and resembled molluscum contagiosum. The lesion on the right side of the nose was an indurated plaque of bluish red color and suggested a fixed eruption. Three months ago the iritis recurred. Two weeks ago, the small nodules were observed to be darker, and a few appeared to be of an apple jelly color on diascopic examination.

A biopsy revealed a "granulomatous reaction suggesting sarcoid." Roentgenograms of the bones of the hands were normal. Roentgenograms of the chest revealed enlarged hilar nodes of the left lung, with a few dense minute nodules along the bronchi. There was some irregular enlargement of the right hilar nodes with peribronchial fibrosis. There were a generalized irregular increase of the bronchial markings on the right and one or two pulmonary infiltrations in the upper lobe of the right lung, laterally. All the pulmonary changes were interpreted as being "possibly tuberculous, probably inactive." The hilar changes were "questionably due to early sarcoid involvement." The Hinton reaction of the blood was negative. The hemoglobin content was 85 per cent, erythrocyes numbered 5,300,000 and leukocytes 7,800. Reactions to tuberculin (old tuberculin, diluted 1:100,000 and 1:10,000) were negative. Reaction to a tuberculin patch test (Vollmer) was negative.

DISCUSSION

DR. C. GUY LANE, Boston: As I looked at this patient, I thought that the eruption was most probably sarcoid. I did not think that it was lupus erythematosus.

DR. G. MARSHALL CRAWFORD, Brookline, Mass.: Clinically, I favor a diagnosis of tuberculosis miliaris facei, and among the dermatologists with whom I have discussed the slide, it was agreed that it appears to be

...ast. The pathologic diagnosis was reported as sarcoid, however, by Dr. Shields Warren. Favoring this diagnosis are the negative tuberculin reactions. From the roentgenograms the hilar changes can be considered early sarcoid. It is hard to say definitely at this point which disease she has. The large lesion is certainly more characteristic of sarcoid.

The referring ophthalmologist has been waiting for me to establish a diagnosis of sarcoid. He cited 1 patient seen in our clinic for whom he made a diagnosis of sarcoid on the basis of a particular type of iritis ten years before we confirmed it.

Basal Cell Epithelioma. Presented by Dr. John G. Downing, Boston.

P. P., a white man aged 33, a machinist, presented a lesion of seven years' duration on the right lower eyelid. At the onset the lesion was marked merely by irritation. Then gradually an ulceration appeared, which has destroyed two thirds of the lower eyelid and has caused separation of the tarsal plate.

The Hinton reaction of the blood was negative. A biopsy revealed basal cell epithelioma.

DISCUSSION

Dr. John G. Downing, Boston: I think the main thing to bring out in this case is that this man has been going to physicians for seven years and he is steadily losing his eyelid. Nobody did anything for him except prescribe an ointment. I saw him once and immediately referred him to the Massachusetts Eye and Ear Infirmary. They performed a biopsy. Dr. Frederick Verhoeff and another pathologist confirmed the diagnosis of epithelioma.

Dr. Jacob H. Swartz, Boston: I had a similar experience with a patient I saw in consultation. The patient had been treated for two years with ointments. Also an ophthalmologist treated the tear duct. Finally a dermatologist questioned the diagnosis. A biopsy was made, and a diagnosis of epithelioma was established. The lesion had extended to the conjunctivas and had eroded a portion of the lower eyelid. In my case the treatment was roentgen irradiation. The surgeons felt that excision was out of the question. I saw the patient two months after irradiation, and the result was beautiful. There was one little point that might have been residual.

A Case for Diagnosis (Sarcoid of Boeck?). Presented by Dr. E. Lawrence Oliver and Dr. Austin W. Cheever, Boston.

J. D., a white American school boy aged 12, presented a lesion in and above the right eyebrow of seven months' duration. He first noted a painful area about the size of a dime. This lesion has slowly grown paler but has increased in size and has become elevated. Loss of hair in the involved portion of the eyebrow was noted at the time the lesion first appeared.

The lesion is a slightly raised circular disk of practically the same color as normal skin encroaching on the eyebrow.

No biopsy has been performed.

Treatment has comprised use of a crude coal tar paste and five small fractional doses of unfiltered roentgen rays. The lesion has slowly increased in size.

DISCUSSION

Dr. Jacob H. Swartz, Boston: I am going to offer the diagnosis of granuloma annulare. The border felt rather firm; the color was not typical, but I thought it was waxy. I think a biopsy is indicated.

Dr. Austin W. Cheever, Boston: I have hesitated to perform a biopsy because of the location of the lesion. I suppose it is the only thing to do. The family have objected. Dr. Oliver favored the diagnosis of granuloma annulare. I wish some one would offer a suggestion of what to do. I have given nine treatments of about 30 r each. No blood count was made for this patient.

Dr. G. Marshall Crawford, Brookline, Mass.: There are case reports of occurrences of mycosis fungoides and Hodgkin's disease with initial lesions on the scalp or face. I have never seen such cases. One might include these possibilities in the differential diagnosis.

NEW YORK ACADEMY OF MEDICINE, SECTION OF DERMATOLOGY AND SYPHILIS

David Bloom, M.D., *Chairman*

George M. Lewis, M.D., *Secretary*

March 7, 1944

A Case for Diagnosis (Perifolliculitis Capitis Abscedens et Suffodiens; Malignant Papillomas?). Presented by Dr. Rudolph Reudemann Jr.

H. F., a white man aged 43, was admitted to the outpatient department of the Albany Hospital on June 23, 1943, for examination of an eruption involving the scalp. Seven brothers and two sisters are living and well, and there is no familial history of cancer or tuberculosis. The patient says that he has not had gonorrhea or syphilis. His general health has always been excellent, and he was a hard worker up to 1940.

The patient states that for a number of years prior to 1917 he had a wart on the top of his head. The wart was about ½ inch (1 cm.) long and had a base about ¼ inch (0.5 cm.) across. In 1917, while he was a private in the Army, a barber at Fort Ontario accidentally cut off the wart. It began to grow slowly, and by the fall of 1940 it had reached approximately 1½ inches (3.8 cm.) in diameter and was elevated about ½ inch. In November 1940, while at work, he struck his head on a pipe and knocked "a hunk off." It bled profusely, and the open area became infected and was surrounded by a painful, fluctuant swelling. It was treated by a local physician until May 1941, a cauliflower-like growth arising about the opening in the meantime. In May 1941 he reported to the Glens Falls hospital, where puncture stabs were made in fluctuating areas and through and through drains inserted. There was a greenish yellow malodorous purulent discharge which grew staphylococci on culture. Wherever puncture wounds were made, new growths immediately appeared with small boils developing beyond them; as they were opened, similar growths developed. The infection extended under the scalp in all directions. Pathologic examination of tissue from these growths was diagnosed as benign papilloma.

The patient was admitted to Albany Hospital on July 6, 1943. Wassermann and Kahn reactions of the blood were repeatedly negative. Cultures from the discharge showed abundant growth of Proteus vulgaris, with no staphylococci, streptococci or fungi. Examination of the blood revealed 5,200,000 red cells and a hemoglobin content of 97 per cent; there were

11,150 white cells, with 74 per cent polymorphonuclear leukocytes, 25 per cent lymphocytes, 1 per cent myelocytes and no eosinophils. Results of subsequent examinations have been essentially the same.

The histologic reports were as follows: (1) keratinizing epithelium resting on edematous, vascular, chronically inflamed fibrous tissue; (2) vascular papilloma of the skin with hyperplasia and keratinization, and (3) the opinion of Dr. F. D. Weidman given Nov. 15, 1943: "When I first looked at the photographs of H. F., I thought of so-called endothelioma capitis, but this is an epithelial tumor. Of course, a study of the sections speedily disposes of the first idea and confirms what was thought of clinically—'warts running wild.' The picture in the sections is that of a papilloma rather than of verruca vulgaris, because the hyperkeratosis which dominates the picture in verruca vulgaris is replaced here by parakeratosis and because in some of the sections, at least, the epidermis is not particularly acanthotic. Indeed, in some places it is thin and yet some long lobules extend from it. At the same time, it is not a simple papilloma, because the cells at the basement membrane are of such highly active type—carcinomatous at some places. It is strange that in these circumstances they remain so well defined by the basement membrane; I am wondering whether they would be found to be really infiltrating, cancerwise, if the biopsy specimens had extended right down to the bone. I am reminded of the cancers that occur at the base of the urinary bladder, which likewise are papillomatous at first and which produce damage by local destruction. Every now and then in general pathology one finds a contradistinction between the histologic structure and the clinical behavior of a tumor, and I feel that this case is just one more instance. The old 'adenoma malignum' of the wall of the stomach is an example. I should say that this case is one of malignant papilloma, paradoxic, though that term is."

The patient was discharged from the hospital with the condition slightly improved on Sept. 21, 1943. After various forms of treatment there were less swelling and tenderness, but a new lesion formed at the site of the biopsy wound. The patient returned on Jan. 25, 1944, for further study. There were more swelling and tenderness, and the papillomatous lesions were more pronounced.

DISCUSSION

DR. FRANK C. COMBES: The history in this case suggests the presence of an infectious process, probably perifolliculitis abscedens et suffodiens. There are several unusual factors: first, the proliferative or vegetative lesions, not unlike those observed in dermatitis vegetans; second, the keratotic lesions, which suggest irritation of the prickle cell layer of the epidermis. In many areas there are lesions resembling a malignant papillomatosis. I think that that is the probable diagnosis.

DR. CHARLES WOLF: I just saw the photograph of this patient, and it reminded me of a patient shown here many years ago, and it might be well to suggest a diagnosis of granuloma coccidioid. I suggest that studies be made in that direction.

DR. ISADORE ROSEN: It is impossible to make a clinical diagnosis from one examination. Some features fit in with perifolliculitis abscedens et suffodiens, with the exception of the secondary vegetating lesions. With regard to the treatment, it might be worth while to try the use of wet dressings of gramicidin in a dilution of 1500. If this does not give the desired result, I suggest more drastic measures, such as thorough desiccation, to be followed by the application of antiseptic solutions.

DR. JACK WOLF: Malignant papilloma should not be overlooked. Papillomas of the bladder frequently become malignant. I agree with the opinion that heroic treatment should be employed.

DR. GEORGE C. ANDREWS: This appears to me to be a classic case of turban tumor. Such tumors become ulcerated and infected. The histologic characteristics of turban tumors vary greatly, and treatment is a difficult problem. They are not particularly radiosensitive.

DR. RUDOLPH REUDEMANN JR.: I considered all the diagnoses suggested this evening, but could not reconcile these verrucous growths with some of them. I felt that one was dealing definitely with an autoinoculable disease. Those "boils" that developed were definitely pyogenic satellite lesions, which were opened and drained. The verrucous growths developed along the line of incision, suggesting vegetating pyoderma. It is my intention to try heroic treatment.

A Case for Diagnosis (Lymphoblastoma?). Presented by DR. A. BENSON CANNON.

J. E. C., a retired policeman, is presented from Vanderbilt Clinic, with lesions of eighteen years' duration. About eighteen years ago, he noted an enlargement in the right upper quadrant of the abdomen similar to those now present. The original lesion left a faint brownish discoloration without scarring. Shortly after the first, another large lesion appeared near the midline over the xiphoid process, and it has been present continuously for the past eighteen years. About four years ago a lesion about the size of a half-dollar appeared on the right side of the chest. This had begun to undergo spontaneous resolution when it was removed for biopsy. There is no familial history of tuberculosis or other relevant disease.

The Wassermann reaction of the blood was negative, and the urine was normal except for a faint trace of albumin. A blood count showed a hemoglobin content of 83 per cent, 4,890,000 red cells and a color index of 0.86. There were 11,100 white blood cells, with 8 per cent small leukocytes, 85 per cent neutrophils, 4 per cent band forms and 3 per cent mononuclears and a platelet count of 162,000.

Roentgenograms of the skull, chest, spine and pelvic bones showed essentially normal conditions except for a questionable fracture of the right first rib. The patient refused to submit to a sternal puncture.

The biopsy specimen was reported as showing leukoblastoma cutis circumscriptum.

DISCUSSION

DR. FRED WISE: Despite the history of eighteen years' duration of a probable leukocytic disease, the tumors resemble nothing more closely than localized lymphoblastomas. The only alternative diagnosis that comes to my mind is a variety of dermatofibroma.

DR. E. WILLIAM ABRAMOWITZ: I cannot add anything definite to the diagnosis, but I should like to be permitted to recount my experience with a physician who had a similar eruption on his back for over twenty years. He had seen several dermatologists, who made various diagnoses. My associates and I thought that he had some form of lymphoblastoma of a benign type. I later heard that he had gone to another dermatologist and received roentgen ray therapy and that the eruption subsided partially. This was about five years ago. About two weeks ago I saw him again, and he said: "I still have this thing. What do you think I ought to do about it?"

DR. ADOLPH ROSTENBERG: This case reminds me of the case of a patient I showed several years ago. Lymphoblastoma was suggested; the man was given

roentgen ray treatment, and the tumor disappeared in a short time. If this patient were given roentgen ray therapy and the tumor disappeared or got smaller, it would favor the diagnosis of lymphoblastoma. The tumor might recur, of course. In my patient aleukemic leukemia developed.

DR. A. RENSON CANNON: I am of the opinion that the patient has a sarcoma and the ultimate termination will probably be fatal. While he has had the eruption for eighteen years, the lesions have become more numerous and much larger and are beginning to become a real hazard to him, although he is physically in good health. I saw a patient with similar lesions many years ago; they were diagnosed clinically and histologically as a Spiegler-Fendt type of sarcoid. Several years later the patient was admitted to the dermatologic service of the City Hospital, where he died of a metastatic sarcoma.

A Case for **Diagnosis (Dermatitis Seborrheica; Drug Eruption Due to Phenolphthalein; Arsenical Dermatitis?).** Presented by DR. WILLIAM CURTH.

R. B., a Negro woman aged 23, is presented from Vanderbilt Clinic, with an eruption involving the forehead, ears, neck and trunk, of six months' duration.

A routine Wassermann test in May 1943 elicited a 4 plus reaction. Results of a physical examination at that time were completely normal, and the disease was classified as latent syphilis (early?). Antisyphilitic treatment was begun on June 16, 1943, with one injection of a bismuth preparation followed by 0.2 Gm. of neoarsphenamine one week later. The next two injections of the neoarsphenamine (0.2 and 0.3 Gm. given at weekly intervals) were followed immediately by chills and fever. Since then the patient has not received any more of the drug. She was given weekly injections of 1 cc. of bismuth subsalicylate, which were continued until Sept. 22, 1943. At that time an eruption appeared, at first on the forehead, gradually involving the ears and neck and finally the trunk. Since October the patient has received fourteen injections of sodium thiosulfate with only temporary effect on the dermatitis. The patient has been in the habit of taking three phenolphthalein (Ex-Lax) pills every three or four weeks regularly since the age of 19. Some pills were taken in Nov. 3, 1943, and when the patient was seen three days later the dermatitis of the forehead was more irritated and showed oozing. Three pills were taken again on Jan. 3, 1944, and two days later there was a change; two weeks later the areas on the face and body began to get much worse. Two roentgen ray treatments given to the face during November 1943 were without effect.

The patient now presents what appears to be a seborrheic dermatitis of the forehead, temples, ears and retroauricular folds. There are also sharply defined, coin-sized to palm-sized, round and oval scaling patches on the sternum, in the axillas, below the left breast, on the abdomen and on the entire back. Some of these patches are oozing, and others are pigmented. The arrangement of some of the lesions resembles that of pityriasis rosea.

Biopsy of an inflammatory lesion of the skin showed exfoliation of the keratin layer where there was evidence of parakeratosis. Edema of the epidermis resulted in the formation of a few small superficial vesicles. Acanthosis was pronounced, with formation of long rete pegs. The cutis was edematous and showed evidence of a chronic inflammatory reaction. The papillae were large and contained dilated capillaries. The lesion was psoriasiform. The diagnosis was chronic psoriasiform dermatitis (seborrheic?).

The Wassermann reaction of the blood in November 1943 was 3 plus. The urine was negative for albumin and sugar.

DISCUSSION

DR. FRANK C. COMBES: I think that one should be able to eliminate seborrheic dermatitis as a diagnosis, since this disease responds to roentgen ray therapy promptly. A drug eruption is the more plausible diagnosis. Although the fixed eruptions due to arsenicals and phenolphthalein not infrequently resemble each other, I favor arsenic as the most likely agent in this case.

DR. ANTHONY C. CIPOLLARO: I think that this is another of the cases in which it is difficult to make a definite diagnosis. I believe that if these lesions were on a white person, the features of psoriasis would be more pronounced. Please note that the histologic diagnosis was chronic psoriasiform dermatitis.

Rosacea-Like Tuberculid (Lewandowsky). Presented by DR. ANTHONY C. CIPOLLARO.

M. J., a woman aged 25, came to the Skin and Cancer Unit of the New York Post-Graduate Medical School and Hospital on Feb. 9, 1944, with an eruption on the face and a severe conjunctivitis of three and a half years' duration.

During this time the patient has been treated for a severe conjunctivitis with autogenous vaccines, vitamins and sulfonamide drugs. Thorough general examination, including that of the blood serum, tests for allergy and roentgenograms of the teeth, did not reveal any abnormalities. Diseased tonsils were removed about eight months ago. From October to December 1943, the patient was given sulfathiazole ointment for application to the eyes, without benefit.

On both cheeks, but more on the right one, and on the chin and the nose there are a number of erythematous, lentil-sized and smaller papules and pustules. The conjunctivas show pronounced injection around the cornea, and the patient suffers from photophobia.

Biopsy of a lesion on the cheek was reported as showing the features of rosacea-like tuberculid of Lewandowsky. The reaction to tuberculin was positive with a dilution of 1:1,000 but negative with a dilution of 1:10,000. A blood count revealed moderate hypochromia and an elevation of leukocytes to 13,500 white blood cells.

Sarcoid. Presented by DR. FRANK VERO.

M. C., a white woman aged 30, is presented from Vanderbilt Clinic, with an eruption on the face and neck of four or five months' duration.

The patient was first seen on Dec. 23, 1943, complaining of an eruption of several weeks' duration which was resistant to all local treatments. There was a history of frequent colds and bronchial ailments.

On both cheeks and on the neck are a large number of solitary and grouped, reddish-brownish, papular nodular lesions, from the size of a pinpoint to that of a pinhead. In some areas an erythematous vesicular papular eruption is present. The lesions on the chin and neck remain unchanged under pressure with a glass, yellowish specks showing in some.

Tuberculin tests elicited negative reactions with dilutions of 1:1,000,000 and 1:10,000. The blood count showed a hemoglobin content of 97 per cent, 5,100,000 red cells and 8,000 white cells, with 69 per cent poly-

morphonuclear leukocytes, 28 per cent small lymphocytes and 3 per cent eosinophils.

Roentgenologic examination of the chest revealed some calcific changes in both hilar regions. Roentgenologic examination of the hands showed no evidence of abnormality in the bones.

A biopsy disclosed an epidermis which was slightly edematous and in one area atrophic. The corium was the site of a diffuse chronic inflammatory reaction. Here and there within it were collections of epithelioid cells. In relation to one hair follicle these formed fairly typical tubercles which contained giant cells. The lesion was evidently a tuberculid.

The patient at first had a superimposed dermatitis venenata which cleared with soothing local medication. She has been receiving ultraviolet irradiation to the face.

Sarcoid; Dermatitis Venenata. Presented by DR. FRANK VERO.

H. W., a white woman aged 21, is presented from Vanderbilt Clinic, with an eruption on the face of twenty-two months' duration.

The patient was first seen on May 18, 1943, complaining of a persistent eruption on the face which had begun about a year previously with small "pimples," slight itching and a sensation of flushing. For several years she has had anemia, for which she has received various preparations of iron; otherwise her health has been good.

Examination at that time revealed on the face (especially on the cheeks, chin and submental region) a large number of isolated and closely grouped reddish-brownish, infiltrated papular and nodular lesions, ranging from the size of a pinhead to that of a small millet seed. On diascopic pressure, the lesions showed minute yellowish discolorations. There was a considerable flush on both cheeks.

Blood counts showed the hemoglobin content to vary between 71 and 78 per cent, with 4,170,000 red cells and a color index of 1; there were 6,150 white cells, with 49 per cent polymorphonuclear leukocytes, 2 per cent staff cells, 22 per cent small lymphocytes, 24 per cent large lymphocytes and 3 per cent transitional cells. Urinalysis and roentgenologic examination of the chest revealed normal conditions. The tuberculin test elicited negative reactions with dilutions of 1:100,000 and 1:10,000.

Biopsy performed by Dr. Machacek was reported to disclose a rosacea-like tuberculid of Lewandowsky or miliary lupoid.

The patient received local and generalized ultraviolet irradiations and injections of liver extract from May to September, when the eruption appeared to be practically cleared. She was seen again on October 21 with an acute, macular, papular and purpuric eruption on the face, which was considered to be due to the intake of anacin (a preparation of acetophenetidin with acetylsalicylic acid and caffeine). This eruption cleared in two or three weeks. She was seen again on February 16, because of an acute dermatitis of the face which was probably due to nail polish. Results of patch tests with two brands (Revlon and Miraglow) were positive. At present the dermatitis is clearing, but she still shows a few small miliary lupoid lesions on the chin.

DISCUSSION OF THE PRECEDING THREE CASES

DR. E. WILLIAM ABRAMOWITZ: Dr. Cipollaro's case does not strike me as one of Lewandowsky's tuberculid. I think that this is a disease which is hard to define except when clinical and histologic appearances coincide. I shall call Dr. Vero's case clinically a case of Lewandowsky's tuberculid and Dr. Cipollaro's case possibly an instance of sarcoid.

DR. ANTHONY C. CIPOLLARO: It is possible that the changes in the eye and the other changes are not related. I think that the same tuberculous process is manifest in the eyes and in the skin.

DR. FRANK VERO: The first patient, the young girl, I saw for the first time last May, when the eruption was of about a year's duration. While she complained of flushing of the face, I could see only small papules and at no time any pustules. Her eruption has practically cleared with ultraviolet irradiation and injections of liver extract. Some of the changes seen at present I consider to be due to a contact dermatitis caused by nail polish.

The second patient's eruption persisted for two months, and when first seen she had a superimposed dermatitis venenata from various medicines.

I believe that the two eruptions belong in the group of miliary sarcoid. Tuberculin tests elicited negative reactions in both patients, whereas persons with rosacea-like tuberculid, according to Sulzberger, Wile and others, are usually hyperallergic to tuberculin, even in high dilutions.

Eruption Due to Sulfathiazole. Presented by DR. E. WILLIAM ABRAMOWITZ.

A. de A., a girl aged 17, came to the Skin and Cancer Unit of the New York Post-Graduate Medical School and Hospital on Feb. 21, 1944, with a generalized eruption of about two months' duration.

About Christmas 1943, the patient had impetigo of the face, for which a physician prescribed a 5 per cent sulfathiazole ointment. When after a few days the eruption became worse, the physician prescribed in addition sulfathiazole tablets to be taken internally, 1 Gm. every four hours. After seven days of this medication, the eruption was worse and the patient complained of sleepiness. The oral administration of sulfathiazole was discontinued, but the use of sulfathiazole ointment was continued, to be applied at the advice of the nurse. After another physician recommended discontinuation of the use of sulfathiazole ointment, the eruption began to improve.

There is an erythematous, crusted, scaly eruption in some areas oozing, on the face, neck, chest, forearms and thighs. The patient complains of moderate pruritus.

The urinalysis gave normal results.

DISCUSSION

DR. E. WILLIAM ABRAMOWITZ: Judging from experience, I should say that this type of eruption continues to spread long after use of the drug (usually externally) is discontinued, and it is extremely resistant to treatment. It assumes the aspect of an infectious eczematoid dermatitis. It is not unusual for an eruption of this kind to last four to six months or even more. It is the duty of dermatologists to warn general practitioners against using this ointment, especially when other measures can be just as effective.

Keratosis Palmaris et Plantaris. Presented by DR. RICHARD J. KELLY.

S. O., a white housewife aged 60, was first seen at Vanderbilt Clinic on Jan. 31, 1944. At that time she complained of thickening, scaling and fissuring of the palms and soles of twelve months' duration. The general health was good, the blood count normal and

logic reactions negative. The blood arsenic level was (.05 mg. per hundred cubic centimeters of dried blood.

The patient had previously been treated with roentgen ray therapy on twelve occasions, with no appreciable effect.

DISCUSSION

DR. A. BENSON CANNON: The uniform thickening of the palms and soles accompanied with little or no inflammation made one think that probably the cause of the trouble was arsenical hyperkeratosis. While keratosis palmaris et plantaris of the acquired type seemed to be the best diagnosis when the patient was first seen, I shall be much interested in the results of treatment directed toward the elimination of the arsenic.

DR. CHARLES WOLF: The pronounced keratosis of the soles with thickening of the nails and the increased amount of detritus subungually lead me to believe that the disease is of much longer duration than one year. However, other characteristics give a clue to a probable diagnosis. There are sharply demarcated borders. There is one real characteristic that this patient has—stippling of the nails. It seems to me that the eruption is psoriasis. I think that if this patient is carefully examined, a small area of psoriasis will be found.

DR. FRED WISE: I agree with the last speaker that a diagnosis of psoriasis should be seriously considered. It is difficult to make a diagnosis of palmar and plantar psoriasis when there are no lesions on other parts of the body. The stippling of the nails is also suggestive. I wonder whether keratoderma climactericum should not be taken into consideration. Textbooks suggest that more or less isolated lesions of rather limited extent are characteristic of this disease, but that does not exclude the possibility that in some cases the lesions become widespread and uniform. The patient might be given estrogenic therapy, in an attempt to find out whether this has any beneficial effect on the keratoses.

DR. A. BENSON CANNON: I believe that Dr. Kelly considered the possibility of psoriasis as well as of arsenical hyperkeratosis. There has been no redness or scaling at any time that would suggest psoriasis. Furthermore, it is not usual to have psoriasis uniformly involving the entire palms and palmar surfaces of the fingers and the entire soles without lesions elsewhere. There was no abnormality of any kind noted in the nails.

Diabetic Panniculitis. Presented by DR. GERALD F. MACHACEK.

M. O., a white widow aged 39, is presented from Vanderbilt Clinic complaining of tenderness of the lower aspect of the abdominal wall of five months' duration.

On March 30, 1943, the patient sought treatment for bilateral exophthalmos. She weighed 223 pounds (101 Kg.), had a tremor and an enlarged nodular thyroid and suffered from tachycardia. Her basal metabolic rate was elevated, several readings being as high as +37 per cent. She also suffered from hypertension and diabetes. On Sept. 17, 1943, the patient complained of tenderness of the lower part of the abdomen. She had been receiving 0.1 Gm. of sodium iodide daily for a two week period alternated with a two week rest period. The patient's abdomen is pendulous, and the wrinkled skin is striated. The striae are not livid. Tenderness of the redundant folds of the panniculus is elicited by palpation over indefinite small nodular infiltrations.

Biopsy disclosed a granuloma surrounding necrosis of the fat lobules.

Urinalysis showed dextrose (4 plus). The blood sugar level was as high as 240 mg., and the basal metabolic rate was elevated to +37 per cent. The blood cholesterol level was 281 mg.

After the patient had received a course of treatment with thiouracil (0.2 Gm. three times a day at first and now 0.1 Gm. once a day), examination revealed that the blood sugar level was 95 mg.; the weight, 182 pounds (82.6 Kg.); the blood cholesterol level, 237 mg. and the basal metabolic rate, —3 per cent. Blood counts were within normal limits. The exophthalmos was unimproved; protrusion of the right eye measured 27 mm. and of the left eye 23, at 101.

DISCUSSION

DR. E. WILLIAM ABRAMOWITZ: Is the eruption in any way related to the injections of insulin?

DR. CHARLES WOLF: I should like to ask Dr. Machacek whether he is fairly certain that he is not dealing with necrobiosis lipoidica diabeticorum.

DR. GERALD F. MACHACEK: It is not necrobiosis; in my opinion it is panniculitis. The two diseases vary considerably.

A Case for Diagnosis (Artefact; So-Called Initial Lesion of Pemphigus; Bullous Dyskeratosis Darier). Presented by DR. LOUIS CHARGIN.

J. E., an unmarried salesman aged 40, is presented with a lesion of the scalp of eight months' duration. The patient is in excellent general health.

About eight months ago a "scab" appeared on the left aspect of the scalp at the border of the hair after vigorous and repeated washing of the scalp. There was some associated itching, and the patient admits repeated scratching of the site and repeated removal of the formed crust. In time the lesion increased to its present size.

The lesion is about 2 by 2 inches (5 by 5 cm.) and is circular in outline; it is superficially eroded. The border is not raised or depressed. Slight oozing is present, and some itching is experienced at times. Alongside the patch, in the hairy scalp are some crusts but no denuded areas.

The lesion has failed to heal since its onset, despite a variety of therapeutic measures. Because an artefact was suspected, an occlusive dressing was tried, but it was without benefit. Consideration was then given to the possibility of a superficial type of epithelioma or the initial lesion of pemphigus.

Biopsy revealed some dilated vessels throughout the middle and upper parts of the cutis, and there was a diffuse moderate infiltration of small round and plasma cells. Within the epidermis was a large cavity. For the most part, the papillary bodies were present and capped with a margin of basal cells, and this formed the base of the cavity. Within the cavity were epithelial cells with deep-staining cytoplasm, but the nuclei were retained. The microscopic diagnosis was so-called bullous Darier's disease.

Since bullous Darier's disease is said to respond to roentgen irradiation, this was tried but without result. Then, on the possibility that it might after all be the primary patch of pemphigus, acetarsone was administered. After the patient had taken 24 tablets a scarlatiniform eruption developed and the drug had to be stopped. There was no noticeable effect on the lesion, except that it was perhaps a little drier.

DISCUSSION

DR. WILBERT SACHS: Clinically, I cannot make a diagnosis. From microscopic studies, I was led to re-

port a so-called bullous Darier's disease. This disease has a characteristic picture, and the patient presents all the features of it. This diagnosis is not corroborated clinically, but microscopically that is the picture.

DR. E. WILLIAM ABRAMOWITZ: I recall a patient with an eruption of the same type, who was presented at the Brooklyn Dermatological Society at one of the early meetings. There was considerable discussion at that time as to whether the patient had the bullous type of lupus erythematosus or pemphigus. His eruption later developed into lupus erythematosus of the face. I do not think that this lesion is an artefact.

DR. ANTHONY C. CIPOLLARO: No one can make an unequivocal clinical diagnosis in this case. I have not the least idea what the disease is, but, since the histologic picture suggests the possibility of bullous dyskeratosis, a diagnosis of Bowen's disease might be made. The treatments which this patient has received are usually effective for the other diseases mentioned. I suggest treating one small area with desiccation and curettage.

DR. WILBERT SACHS: There is no dyskeratosis in this lesion. This is a so-called bullous Darier's disease. The cells are large; the nuclei are well stained and round, and the prickles are gone. There is normal epithelium above the areas of so-called dyskeratosis. These cells are ballooned, not dyskeratotic.

DR. MAX SCHEER: I cannot get away from Dr. Chargin's first impression, that the lesion is an artefact, self induced. The patient says that he is a nervous, apprehensive person, has had an impulse to rub and scratch and admits that he traumatized and continues to traumatize the lesions. My impression is that the eruption is an artificially produced dermatitis, and I suggest that Dr. Chargin refer the patient to a psychiatrist.

Riehl's Melanosis. Presented by DR. HELEN CURTH.

I. de B., a Negro woman aged 39, is presented from Vanderbilt Clinic with a pigmentation of the face of six months' duration.

The patient was well until last summer. She has always freely used Noxzema, Pond's vanishing and cold creams and petrolatum as a powder base. There was no undue exposure to the sun, as the patient knew that her skin would peel after such exposure. In August 1943, her face began to become darker. She continued to use the creams until January 1944, when she was first seen in the department of dermatology at the Vanderbilt Clinic. Bleaching creams with hydrogen peroxide and ammonia did not improve the discoloration.

There is reticular hyperpigmentation on the bridge of the nose and on the forehead, with confluent hyperpigmentation on the cheeks, upper lip and chin. (The patient thinks that lately this confluence has been interrupted in places by normal skin.) There is a mild papular rash on the right side of the chin. Further examination reveals pigmented spots on the buccal mucosa, mild keratosis pilaris of the arms and several keloids.

The Wassermann and Kahn reactions of the blood were negative.

Biopsy disclosed hyperkeratosis and a slight acanthosis of the edematous epidermis. The corium showed a striking perivascular infiltration of small mononuclear cells. Just beneath the epidermis were numerous chromatophores. The lesion was a melanodermatitis and was probably the so-called melanodermatitis of Riehl.

DISCUSSION

DR. CHARLES WOLF: The patient is exposed to heat and employs cosmetics; although she disclaims exposure to the sun directly, nevertheless hers is a sharply demarcated, hyperpigmented eruption without any inflammatory clinical manifestations. Riehl's melanosis is about the best diagnosis one can offer at present.

DR. FRED WISE: I think the diagnosis is acceptable under that name. I should like to call attention to a booklet (Pierini, L. E.: Dermatosis pigmentarias de la cara y del cuello. Imprenta de Alfredo Frascoli, Buenos Aires, Argentina, 1941) published in Argentina, containing numerous photographs of dark-complexioned natives, not Negroes, in whom this type of eruption is said to be extremely common after prolonged use of cosmetic creams.

DR. HELEN CURTH: Riehl's melanosis was first described in Europe. It occurs mostly in dark-skinned women under certain conditions. I believe that the histologic changes in this patient are rather characteristic of this disease. There is an increase of the chromatophores, and there is evidence of an inflammatory process in the cutis. Clinically, there are no signs of an inflammatory process.

Systematized Epidermal Nevus. Presented by DR. HELEN CURTH.

C. K., a Jewish girl aged 13, is presented from Vanderbilt Clinic, with generalized dark streaks on the skin which began to appear at 6 months of age.

The patient is the third of four children, and none of the others are similarly affected. There is no consanguinity of the parents and no history of cancer in antecedents.

When the patient was first seen in the Vanderbilt Clinic, in 1937, the eruption was almost as widespread as it is now. Elevated hyperkeratotic thickened brownish to blackish skin is present symmetrically on the neck, in both axillas, on the flexor side of the arms in the cubital areas, on the extensor surface of the elbows and forearms, on the lower sides of the chest on the flanks, in the inguinal areas (assuming a streaklike appearance from the buttocks to the flexor side of the knees and extensively on the extensor surface of the knees), around the ankles and on the dorsa of the feet. There is mild redness of the skin in the axillas. Last Christmas, blisters on the soles were observed which may have been impetigo.

Results of a general examination and analysis of the blood have been repeatedly normal.

Biopsy of specimens from the axillas, taken in June 1937, revealed focal papillary proliferations of the epidermis with pronounced hyperkeratosis and acanthosis. The epidermal cells disclosed a spongy degeneration, particularly among cells which contained keratohyalin. There were no abnormal deposits of melanin. This substance seemed to be depleted rather than increased in quantity. The diagnosis was epidermal linear nevus.

Biopsy from the shoulder in December 1943 disclosed areas of loose hyperkeratosis and some parakeratosis. The epidermis at these sites showed evidence of acanthosis and a peculiar spongiosis of the epidermal cells as well as pronounced disturbance of the keratohyalin. There was also evidence of dyskeratosis in that many of the shrunken epithelial cells were highly eosinophilic. The lesion was interpreted as an epidermal nevus with changes suggesting epidermodysplasia verruciformis.

Treatment has consisted of about forty-six injections of liver extract, roentgen irradiation, exposure to the sun, vitamin A in doses of 50,000 U. S. P. units for nine

weeks and 100,000 units later, with local applications of salicylic acid and chrysarobin. While the patient's mother has thought that there was sometimes definite improvement following the use of these medicaments, I was never convinced of this.

DISCUSSION

DR. HELEN CURTH: Some, but not all, of the features of this verruciform nevus seemed to fit into the diagnosis of acanthosis nigricans, Darier's disease or erythroderma ichthyosiforme congenitale. I thought I had a lead when Dr. Machacek observed vacuolation of the epidermal cells and changes of the keratohyalin which he considered characteristic of epidermodysplasia verruciformis. Weisman and Montgomery (ARCH. DERMAT. & SYPH. **45**:259 [Feb.] 1942) described a case of the same type of nevus in a boy of 11. I believe that the photographs of that patient and the patient presented tonight could be interchanged. This case is again proof that epidermodysplasia verruciformis is an epithelial nevus.

Book Reviews

Manual of Clinical Mycology: Military Medical Manuals. Prepared under the Auspices of the Division of Medical Sciences, National Research Council. Price, $3.50. Pp. 348 with 148 illustrations. Philadelphia: W. B. Saunders Company, 1944.

The newest addition to the study of mycology, in 1944, is a Manual of Clinical Mycology, one of a series of Military Medical Manuals, "developed in order to furnish the medical departments of the United States Army and Navy with compact presentations of necessary information in the field of military medicine." It is the result of cooperative work of the following authors: Norman F. Conant, Donald Stover Martin, David Tillerson Smith, Roger Denio Baker and Jasper Lamar Callaway. The Manual consists of twenty-five chapters and an appendix. The diseases discussed are actinomycosis, blastomycosis, coccidioidomycosis, geotrichosis, chromoblastomycosis, cryptococcosis, moniliasis, histoplasmosis, sporotrichosis, maduromycosis, aspergillosis, penicilliosis, mucormycosis, rhinosporidiosis, the dermatomycoses, piedra, trichomycosis axillaris, tinea versicolor, erythrasma and otomycosis. A special chapter is devoted to the immunology of the dermatomycoses, cutaneous and serologic tests, dermatophytids and desensitization. Another chapter deals with the mycology of the dermatomycoses, the classification of fungi and their morphologic characteristics in lesions and in cultures. The fundamental of elementary mycology, the general classification of fungi and the various forms of myceliums and spores are discussed in a separate chapter. The last chapter is devoted to the description of thirty common contaminants. In the appendix are given mycologic laboratory technics, pathologic and immunologic methods, methods of examination under Wood's light and a formulary of useful prescriptions. The list of references in each chapter includes publications appearing up to 1944. The illustrations are a major asset of the book. There are numerous and instructive clinical photographs, reproductions of roentgenograms of lungs and bones, photomicrographs of fungi in the lesions observed on direct examination or in sections, biopsy or autopsy material and pictures of gross and microscopic morphologic structures of cultures.

The book has been written for general medical practitioners. Three fourths of it is devoted to the discussion of mycotic diseases which are or may become systemic. About one fourth is given to the discussion of the dermatomycoses. The discussion of the systemic mycoses is remarkably complete for a manual of this size, but the description of the dermatomycoses is somewhat too brief and may not satisfy a dermatologist. The expressions "Trichophyton unguium" (page 217) and "Trichophyton corporis" (page 221) should be replaced by the terms "trichophytosis unguium" and "trichophytosis corporis." In the description of tinea capitis it is stated (page 230) that hair is loosened and can be epilated readily. Unfortunately, this is not quite so. If it were so easy to epilate hair manually, then it would be easy to cure tinea capitis by manual epilation without the use of roentgen rays. In cases of tinea capitis, however, only a few hairs can be epilated, that is, removed entirely from their follicles by the manual method. As a rule attempts at manual epilation effect only breaking of the hair, leaving a good part of it in the follicle; that is why the disease continues. The statement that alopecia usually develops in the infected area is somewhat misleading. In cases of ordinary (noninflammatory) tinea capitis there is no true alopecia. The hairs are not absent but are merely broken off short.

The reviewer cannot agree with the statement that favus may be produced by Trichophyton violaceum or Microsporon gypseum. T. violaceum does not cause favus. Lesions produced by this fungus on the scalp have all the characteristics of ringworm and not of favus; namely, short, broken or "black dot" hairs but with no scutula, no atrophy, no alopecia and no "mousy odor," and there is a tendency toward spontaneous cure at the age of puberty. The microscopic morphologic structure of T. violaceum in the hair is that of a typical trichophyton, usually of the endothrix type. The statement that M. gypseum may cause favus is, in this reviewer's opinion, a misunderstanding. If a fungus is a true microsporon, then it will cause ringworm but not favus, and the appearance of the parasite in the hair will be that of a microsporon. All this confusion regarding the cause of favus is due to the classification of the dermatophytes in the Manual, a classification which eliminates the genus Achorion and redistributes several species formerly placed in this genus among trichophytons and microsporons. This is done in order to simplify the matter. In the reviewer's opinion this new classification complicates rather than simplifies it. The earlier classification worked out by Sabouraud and followed by many contemporary mycologists and dermatologists established the genus Achorion and referred to it all fungi capable of producing favic scutula in the human or animal skin, regardless of the gross and microscopic morphology of cultures. According to Sabouraud's classification, favus is an independent clinical and etiologic entity different from ringworm and is due (with very rare exceptions) to the fungus Achorion schonleinii. Other fungi placed in the genera Trichophyton and Microsporon have nothing to do with favus. This conception seems cor-

rect, simple and easy to understand. The matter becomes much more complicated if one assumes (as is done in the Manual) that ringworm and favus are only different clinical manifestations which may be produced by the same fungus and that favus, also, may be caused by T. violaceum or by M. gypseum. The assumption is, in other words, that the same fungus (for instance, T. violaceum) in some cases will produce a typical ringworm of the scalp and in other cases will cause a typical favus with scutula. With this conception the reviewer cannot agree. It would also be advisable to mention in the description of favus that scutula are not simple crusts but masses of fungi in almost pure culture, a pathognomonic feature of favus not present in any other disease of the skin. It would be useful to stress that scutula constitute the best material for a microscopic diagnosis of favus, because the picture of fungi in a scutulum is even more characteristic of favus than the appearance of fungi in a hair.

The description of the appearance of Trichophyton and Microsporon in the hair (pages 246 and 247) is somewhat incomplete and may confuse a laboratory worker. It is stated that a Trichophyton endothrix shows parallel rows of spores inside the hair, a Trichophyton ectothrix shows parallel rows of spores outside the hair and a Microsporon forms a mass of spores around the hair. This is the truth but not the whole truth. It should be added that T. endothrix may show a mass of spores without linear disposition inside the hair, which looks like a sack full of potatoes (a common picture for T. violaceum). T. ectothrix is present not only outside but also inside the hair, and the spores may be arranged not only in chains but also irregularly. The Microsporon gives not only a mass of spores around the hair but filaments inside the hair.

It is stated (page 253) that the species known under the names Trichophyton album, Trichophyton discoides and Trichophyton ochraceum are identical with the fungus Achorion schonleinii of favus. The question was investigated by Sabouraud almost half a century ago, and he concluded that they are not identical. He placed the three species in the genus trichophyton under a group name, Trichophyton faviforme: that is, with cultures resembling the fungus of favus but not identical with it. Several reasons impelled Sabouraud to establish this differentiation. Formation of scutula is usual and a most characteristic feature of favus, in spite of the fact that favus may produce lesions resembling trichophytosis (favus herpeticus). Cultures obtained from favic scutula are not of T. faviforme type but of Achorion schonleinii type. Lesions caused by T. faviforme on human and animal skin have all the features of ringworm and not of favus, and there are no scutula. The microscopic picture of T. faviforme in a hair differs from the picture seen in favus and is of the type T. ectothrix.

On page 319 there is a formula for the preparation of Sabouraud's dextrose agar medium, giving the amount of agar as 3.5 per cent and advising the use of distilled water. The original formula suggested by Sabouraud contained only 1.8 per cent agar, and distilled water was not required. Plain tap water was found to give excellent results.

In the discussion of pleomorphic degeneration (page 249) it would be useful to mention that this can be prevented by making a transfer of the isolated strain on Sabouraud's "conservation" medium, consisting of 3 per cent of peptone and 1.8 per cent agar in plain water without carbohydrates.

Essentials of Allergy. By Leo H. Criep, M.... Price, $5. Pp. 381, with 42 illustrations in black and white and 1 plate in full color. Philadelphia: J. B. Lippincott Company, 1945.

In this compact volume the author has given a large amount of information, which is concisely stated and easily understood. The subject matter is presented in outline form and includes chapters on anaphylaxis, allergy, diagnosis and treatment of allergy, pollen allergy, bronchial asthma, nasal allergy, cutaneous allergy, serum allergy, drug allergy and bacterial allergy. Selective bibliographies supplement each chapter.

Some chapters are followed with case reports, which illustrate essential practical considerations and emphasize diagnostic and therapeutic methods. A good number of photographs and tables are included, which greatly enhance the value of the book.

Useful diets free from egg, wheat and milk are given and numerous recipes which do not contain wheat and egg.

A weak section of the book is the chapter on cutaneous allergy. Not only is the subject poorly presented and inadequately covered, even for a book of this size, but many of the opinions expressed are vague and will not meet with the approval of most dermatologists. For example, in the discussion of pathology and immunology of contact dermatitis (page 218), the following statement appears: "It should be pointed out that the lesion of contact dermatitis may be reproduced either by patch test (that is, by exposing the skin to the causative substance), or (as in the case of poison ivy and some other agents) by ingestion . . . The lesion produced as a result of contact in these individuals is a vesicle."

Contact dermatitis does not produce only one type of lesion (vesicle), nor do positive reactions to patch tests always reproduce clinical lesions. If by "ingestion" the author meant to indicate that contact with an allergen may also be from within, why should he select poison ivy as an illustration?

The last paragraph on page 219 begins with more or less correct observations but ends in meaningless phraseology: "Those substances which are soluble in the oil of the skin, or others which can penetrate the horny layer of the epidermis and have an affinity for the epidermal cells, will more readily produce contact dermatitis. For this reason various chemicals and dyes and the oily fraction of certain plants are good contactants. In this group are found instances of allergy to vegetable and plant oils, such as poison ivy and ragweed, occupational contact dermatitis due to metals and chemicals, similar lesions due to contact with fur dyes, leathers, cosmetics and drugs. The condition is characterized by intense pruritus, which leads to scratching and skin trauma."

On page 231 the following statement appears. "It occasionally becomes necessary to differentiate an allergic dermatosis from such conditions as scabies or seborrheic dermatitis or insect bites. Frequently, one of these two skin conditions is found together with some form of allergic dermatosis. In seborrheic dermatitis one does not elicit any of the criteria for allergic diagnosis. The distribution of the lesion is about the hairy portion of the skin. The skin is oily and acne is present. The lesion is pustular." Most of these statements are dermatologically incorrect.

Many other objections could be cited, but for the sake of space the reviewer will stop at this point and respectfully suggest that in the next edition the author collaborate with a dermatologist.

All in all, the book deserves circulation among medical students and general practitioners, for whom it is intended.

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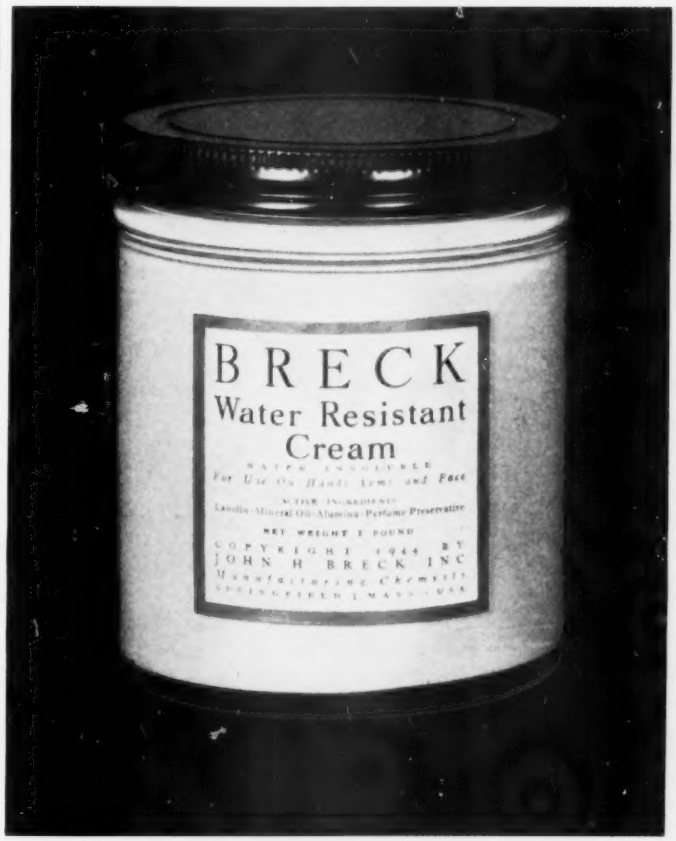

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Unlike the "hay fever" victim who dreads only the approach of the "pollen season"—the patient afflicted with cosmetic allergy is exposed to offending allergens all year round. Unless, of course, her physician assists her in the management of this common sensitivity by prescribing Hypoallergenic Cosmetics of fine quality—such as ALMAY. • The result of years of highly specialized research and experimentation, ALMAY Hypoallergenic Cosmetics include not only the "essential" cosmetics such as lipstick, face powder and rouge, but cold cream, astringent, hand cream, mascara, soap, etc. • To help overcome the difficult problem of *hyperallergic* patients, ALMAY offers Raw Material and Clinical Testing Sets, and cooperates with the physician in developing "personalized" cosmetics for such cases.

Write for FREE copies of "Cosmetic Sensitivity", "Cosmetic Formulary"; and "Beauty Aids by Almay" (for the consumer)

The ALMAY
Raw Material Testing Set and Clinical Testing Set

ALMAY
COSMETICS

ALMAY, INC., 56 COOPER SQUARE. NEW YORK 3, N. Y.
Sole Distributors: **Schieffelin & Co.,** New York 3, N. Y.

This can be your
WIFE'S LUCKY DAY

We've a message—and a gift—for your wife. We want her to try our preparations. It will take about an hour of her time, and in return for that courtesy we will present her with a Beauty Counselor $1.00 lipstick in any shade she selects.

We believe she will enjoy the hour. But, most important, we hope she will tell *you* about our Try-Before-You-Buy method of selection—how each customer has a chance to use these hypo-allergenic preparations, without charge, to see that those recommended really suit her skin.

If you also care to help your office attendant without cost or obligation, we will gladly extend this special offer to her. Please use the coupon below.

Beauty Counselors

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beauty counselors, inc., 17108 Mack Avenue, Grosse Pointe 24, Mich., Dept. AD-2

☐ Send me your private formulas.

☐ Give my wife a $1.00 lipstick and a demonstration. You will find her at:

Street_____ City_____ State_____ Phone_____

☐ Give my attendant a $1.00 lipstick and a demonstration. You will find her at:

Street_____ City_____ State_____ Phone_____

Doctor_____ Address_____

Luzier Cosmetics and Allergy

Women use cosmetics because they have developed a need for them: they are essential to modern standards of good-grooming and therefore contribute to a sense of well-being. Your patient's appearance, viewed cosmetically, is a factor that deserves your consideration both during hospitalization and convalescence. Cosmetics cannot lift faces, but they certainly perform wonders when it comes to lifting a woman's spirits. Women have an instinctive desire to look pretty and to smell sweet.

Since cosmetics are so universally used it is not to be wondered that they sometimes figure in the field of allergy. That is why when there is a history of allergy we suggest that patch tests be made with those of our products the subject is using or contemplates using. If they test positive, further testing with their constituents is indicated to determine the offending agents. These found, we frequently can modify our formulas to suit the subject's requirements. The patch test is generally considered best for testing cosmetics because it most closely approximates the conditions under which they are normally used.

While our products are free from so-called common cosmetic allergens, such as orris root and rice starch, we feel it should be made clear that any of their normally innocuous ingredients might be allergenic to the allergic individual. It is our practice to write our patrons a letter to this effect when a history of allergy is involved.

It is our experience that many persons with allergic constitutions cannot tolerate scented cosmetics; therefore we routinely recommend and select unscented products when there is a history or suspicion of allergy. This practice is not to imply or suggest that the subject is sensitized to perfume; it is solely to safeguard against the possibility.

In specific cases of allergy or suspected allergy, when the subject is using or contemplates using our products, we are pleased on his request to send her doctor the involved raw materials for patch testing, also such information concerning our products as may have a bearing on the case.

Since in the light of present knowledge it is not possible, save in specific cases, to make non-allergenic cosmetics, we believe the cosmetic requirements of the allergic individual should be considered by her doctor in the light of the formulas and general characteristics of the products she is using or contemplates using.

Luzier's, Inc., Makers of Fine Cosmetics & Perfumes

KANSAS CITY, MISSOURI

WHY YOU MUST SAVE ALL KINDS OF PAPER...

Each month, 35,000,000 V-boxes go overseas to the armed forces. It requires 81 tons of supplies a month to send a soldier overseas and these supplies are all wrapped or tagged with paper.

Your corrugated cartons and cardboard boxes can go into blood plasma containers—*if you make sure they're collected.*

Your wastebasket scraps can help supply the needed wrapping and packaging materials—*if you make sure they are collected.*

Blood plasma is needed for treating the thousands of wounded and sick left in the wake of war. Every precious bottle of life-saving blood plasma is wrapped in corrugated paper and boxed in a heavy brown paperboard container.

Civilian use of paper and paperboard products has been seriously curtailed to meet all military demands for these products. Before normal civilian needs can be met again, thousands of tons of waste paper will be needed by the mills.

Your old newspapers can help speed the return of the nation to peacetime pursuits—*if you make sure they're collected.*

Your old magazines and books can help meet present vital needs—*if you make sure they're collected.*

Industry's conversion to peacetime production and the demobilization of the armed forces will require vast supplies of paper.

EVERY SCRAP OF EVERY KIND OF WASTE PAPER IS NEEDED TODAY

U. S. VICTORY WASTE PAPER CAMPAIGN

Benefits of Mild-Soap Care Shown in Clinical Tests

SKIN health is akin to skin beauty. And in advising women on the care of their complexions, dermatologists have always stressed the value of mild soap and water—and of proper daily cleansing.

In developing Camay as a toilet soap especially for the skin, we have devoted painstaking effort, research and time, to making it a soap of exceptional mildness. That Camay does fulfill rigid requirements for mildness has been repeatedly demonstrated in skin studies—patch tests, immersion tests, and other authoritative tests.

Realizing the importance of proper skin care, we have developed a cleansing routine embodying the recommendations of eminent dermatologists. Called the Camay Mild-Soap Diet, it insures proper, twice-a-day cleansing with a truly mild toilet soap.

Recent studies, conducted under exact clinical conditions and supervised by competent dermatologists have further demonstrated the value of this Camay skin-cleansing routine on the skins of over 100 women. Examination showed that the change from their former cleansing methods to the Camay Mild-Soap Diet was beneficial in a very high percentage of cases. In the words of the attending dermatologists, "*Camay is really mild . . . it cleansed without irritation.*"

When you are called on to recommend a mild toilet soap for daily use, we believe that you may recommend Camay with confidence. In addition to its outstanding mildness, Camay lathers abundantly and has a delicate fragrance that women find appealing.

Samples of Camay are available for personal or office use and observation. Address your requests to Procter & Gamble, Cincinnati 1, Ohio.

CAMAY

A product of Procter and Gamble

Jupiter's Headache

Never before had JUPITER suffered with such a headache. In desperation he summoned the gods to Olympus and tried the remedies they suggested but without relief. Unable longer to bear the racking pain, he commanded his son VULCAN to cleave his head with an axe. Swish! the axe fell and out of JUPITER'S head stepped MINERVA, goddess of wisdom, fully grown, clad in shining armor, and chanting a pean of victory. JUPITER, apparently, had an IDEA.

* * * *

We, too, about 10 years had an idea for a better method of cleaning the skin. CREAM OF SOAP* is that method, and with it, inexpert hands can make the skin clean—quickly, thoroughly, harmlessly. There is no lather to develop. CREAM OF SOAP* is already in colloidal solution. It adsorbs the surface soil when rubbed on the skin, then rinses off completely with cold, hot, soft or hard water. CREAM OF SOAP* is neutral, has no perceptible chemical action on the skin surface, and can be safely used even when the skin is sensitive, irritated or disturbed. Samples gladly sent on request. Personal Luxuries Co., 55 West 16th St., New York 11, N. Y.

For a clean scalp..

YOU HAVE undoubtedly told many of your patients that a clean scalp is as important as clean hands . . . that a healthy scalp has much to do with a healthy skin, particularly the skin of the face.

Perhaps you have recommended the use of Packers Tar Soap. As a gentle, pleasant and dependable cleansing agent for the scalp and hair, Packers has won the approval of many dermatologists.

Packers also offers the advantage of economy. Shampoos with this famous cake soap average less than a penny—about one-fourth the cost of bottled shampoos.

PACKERS TAR SOAP, INC.
MYSTIC, CONNECTICUT

Penicillin DOSAGE TABLE*

INDICATIONS	INITIAL DOSE (UNITS)	CONTINUING DOSAGE (UNITS)	UNITS IN 24 HR.	REMARKS
Serious Infections (staphylococcus, clostridium, hemolytic streptococcus, anaerobic streptococcus, pneumococcus, gonococcus, anthrax, meningococcus) Adults and children	15,000 to 20,000	(a) Intravenous drip: 2000 to 5000 every hr.	40,000 to 120,000 or more	(a) Dissolve ½ of 24 hr. dose in 1 liter (1000 cc.) normal saline; let drip at 30 to 40 drops per minute.
		or (b) Intramuscularly: 10,000 to 20,000 every 3 or 4 hr.	40,000 to 120,000 or more	(b) Concentration: 5000 U. per cc. normal saline.
		or (c) Intramuscular drip	40,000 to 120,000 or more	(c) Total daily dose in 250 cc. normal saline.
Infants	5000 to 10,000	3000 to 10,000 intramuscularly every 3 hr.	20,000 to 40,000 or more	Each dose in 1 or 2 cc. of normal saline.
Chronically infected compound injuries, osteomyelitis, etc. Adults and children	5000 to 10,000	10,000 every 2 hr. or 20,000 every 4 hr. intramuscularly or intravenously. Larger doses may be necessary at times.	40,000 to 120,000 or more	Concentration for intramuscular inj.: 5000 U. per cc. normal saline. For intravenous inj.: 1000 to 5000 U. per cc. Supplement with local treatment.
Gonorrhea	20,000 every 3 hr. intramuscularly for 5 doses		100,000	Results of treatment should be controlled by culture of exudate.
Empyema Adults and children	30,000 to 40,000 once or twice daily into empyema cavity		30,000 to 80,000	Dissolve in 20 to 40 cc. normal saline and inject into empyema cavity after aspiration of pus.
Meningitis Adults and children	10,000 once or twice daily into subarachnoid space or intracisternally		10,000 to 20,000	Concentration: 1000 U. per cc. normal saline.
Bacterial Endocarditis Adults and children	25,000 to 40,000	25,000 to 40,000 every 3 hr. intramuscularly	200,000 to 300,000	Continuous treatment for 3 weeks or longer. In a few cases the intravenous drip is more advantageous.

*Based upon recommendations by Chester S. Keefer, *War Production Board Penicillin Leaflet*, Apr. 1, 1945; and by Wallace E. Herrell and Roger L. J. Kennedy, *Journal of Pediatrics*, 25:505, Dec., 1944.

● *Write for pocket size copies of this Dosage Table*

ACCEPTED AMERICAN MEDICAL ASSN. Council on Pharmacy and Chemistry

Penicillin Sodium–Winthrop is available in vials (with rubber diaphragm stopper) of 100,000 Oxford Units.

WINTHROP CHEMICAL COMPANY, INC.

Pharmaceuticals of merit for the physician

NEW YORK 13, N. Y. WINDSOR, ONT.